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VAN DIE REDAKSIE

DIE BEHANDELING VAN AKUTE BARBITURAAT-VERGIFTIGING

Die behandeling van barbituraat-vergiftiging is 'n belangrike mediese noodtoestand. Die toestand mag met reg 'n siekte van die beskawing genoem word, want dit is byna uitsluitlik tot die hoër inkomstegroepe van die samelewing, en in Suid-Afrika, tot die blanke gedeelte van die bevolking, beperk. Ondervinding het getoon dat dit met die seisoene saamgaan, aangesien dit meer dikwels in die warm, klam maande van die jaar aangetref word. Ondervinding het gewys dat daar min gevalle as gevolg van 'n oordosis, wat per ongeluk geneem is, voorkom; in die oorgrote meerderheid is die pasiënte se bedoeling om selfmoord te pleeg.

Die mees onmiddellike en belangrike gevaar wat dit vir die lewe inhou, is die asemhalingsbelemmering wat deur die sedatiewe werking van die middels op die sentrale senuweestelsel veroorsaak word. Asemhaling word swak en vlak, wat tot ophoping van koolsuurgas in die bloedstroom lei, en wat op sigself 'n narkotiese uitwerking het. Die oligurie en anurie wat voorkom, word heelwaarskynlik deur skok veroorsaak, terwyl die afwesige reflekse die algemene bedruktheid van die senuweestelsel weerspieël. Die behoudende benadering tot behandeling is soortgelyk met dié wat na narkose toegepas word: onbelemmerde luggang, sorgvuldige waghou vir terugvloei van maaginhoud met herstel van die keelreflekse, brakings, ens. Maagspoeling is afgekeur as 'n prosedure wat moontlik tot verslukpneumonie kan lei, maar daar bestaan geen twyfel dat die bewussyn nie herwin word voordat die verdowingsmiddel uit die liggaam verwyder is nie. (Om hierdie rede is selfs ‚omruilingsoortapping' as 'n uiterste lewensreddende maatstaf aan die hand gegee.[1]) Die bloed-barbituraat hoogte is 'n goeie aanduiding van die ernstigheid van die geval en—as die laboratoriumskattings minder omslagtig was—sou dit moontlik ook 'n goeie praktiese aanwysing vir prognose gewees het. Vanuit 'n praktiese oogpunt rus alles aanvanklik op die mate van asemhalingsbelemmering wat aanwesig is, en die uitslag sal grotendeels afhang van die sukses waarmee dit behandel kan word. Die meeste klinici huiwer nie om sentrale senuweeprikkelmiddels, soos bv. leptasol, pikrotoksien of amfetamien, te gebruik nie, terwyl andere meer

EDITORIAL

THE TREATMENT OF ACUTE BARBITURATE POISONING

The treatment of barbiturate poisoning is an important medical emergency. The condition may rightly be called a disease of civilization, for it is almost exclusively confined to the higher income groups of the community and in South Africa to the European section of the population. Experience has shown it to be seasonal, being more frequently encountered in the hot, humid months of the year. Experience shows that few cases follow accidental overdosage; in the vast majority the patients intend to commit suicide.

The most immediate and important danger to life is the respiratory impairment caused by the depressant action of the drugs upon the central nervous system. Respirations become weak and shallow, leading to accumulation of carbon dioxide in the blood stream, which itself has a narcotic effect. The oliguria or anuria which occurs is probably caused by shock, whilst the absent reflexes and deep coma reflect the general depression of the nervous system. The conservative approach to treatment is similar to the post-anaesthesia regimen: unobstructed airway, careful watching for regurgitation with recovery of the throat reflexes, vomiting, and so on. Gastric lavage has been condemned as a procedure likely to lead to aspiration pneumonia, but there is no doubt that the return of consciousness does not occur until the drug has been eliminated from the body. (For this reason, even 'exchange transfusion' has been put forward as an extreme life-saving measure.[1]) The blood-barbiturate level is a good index of the severity of the case and—if the laboratory estimation were less cumbersome—would probably be a good practical pointer towards the prognosis as well. From a practical point of view everything hinges initially upon the degree of respiratory impairment that is present, and the outcome will depend for the most part upon the success with which it can be treated. Most clinicians do not hesitate to use central nervous stimulants such as leptazol, picrotoxin or amphetamine, whilst others are more cautious over the place of these drugs in

versigtig is oor die rol wat hierdie middels by behandeling speel. Selfs matige oordosisse van hierdie middels het braking, oormatige krampe (selfs stuiptrekkings), hoë temperatuurverhoging en 'n verhoogde bloeddruk as gevolg; en in ernstige gevalle het hulle nie hulle waarde as lewensreddende middels bewys nie.

Twee jaar gelede het Shaw *et al.*[2] gerapporteer dat 'n middel, wat hulle besig was om te ondersoek en wat sederdien bemegried (Megimide) genoem is, 'n beslis teenstrydige uitwerking op barbiturate' by jong rotte met opgewekte akute barbituraat-vergiftiging, gehad het. Hulle het daarop aanspraak gemaak dat die middel by hierdie diere die periode van koma verkort het en die bewussyn gouer laat herwin het. Daarna is verskeie suksesvolle reekse van menslike gevalle gerapporteer. Shulman *et al.*[3] het gevind dat gevalle wat in koma was, tot 'n ,veilige toestand' gebring kon word—d.i. 'n staat van bewussyn wat amper soos ligte verdowing is—deur herhaalde inspuitings van bemegried wat met tussenposes van 5 minute binneaars toegedien word. Alhoewel die presiese aard van Shulman se ,veilige toestand' skerp kritiek uitgelok het weens die vaagheid daarvan, bestaan daar min twyfel dat bemegried die lewens van ontelbare persone, wat andersins weens akute barbituraatvergiftiging sou gesterf het, gered het.

Terselfdertyd staaf die res van die bewyse nie die ander aansprake wat Shaw en sy kollegas vir die middel maak nie. Eerstens verkort bemegried nie die tydperk van bewusteloosheid nie, alhoewel 'n onmiddellike uitwerking van 'n inspuiting is om die pasiënt tydelik ,op te wek'. Hierdie eienskap is onlangs deur 'n Deense kliniese span in Kopenhagen bevestig; binne 'n halfuur na 'n binneaarse inspuiting van bemegried, het 22 diep bewustelose pasiënte op sterk prikkelings gereageer en getoon dat daar reflekse was—,but in most cases the patients lapsed into coma after the first infusion and a further infusion had to be given'.[4] Dit is dus duidelik dat hierdie eienskap van die middel moontlik lewensreddend is, soos Clemmesen (van dieselfde Deense span) by 7 gevalle van asemhalingsverlamming met algehele apnee weens akute barbituraat-vergiftiging, getoon het. Al 7 gevalle het kortlik maar krities op die middel gereageer.[5] Shaw se tweede aanspraak—dat bemegried 'n direkte farmaseutiese teenstander van die barbiturate is—skyn ook ongegrond te wees. Dit lyk of dit baie eerder 'n onspesifieke sentrale senuweeprikkelmiddel, soos bv. pikrotoksien, is, aangesien die aanwending daarvan refleks-hiperaktiwiteit, elektro-ensefelografiese veranderinge en self psigotiese ongesteldhede kan veroorsaak. Uit 50 pasiënte van die Kopenhagen reeks, het 15 'n psigotiese toestand, wat deur ylhoofdigheid en hallusinasies gekenmerk is, ontwikkel. Nege van hierdie pasiënte was aan barbiturate verslaaf, en dit word heel voorlopig gesuggereer dat bemegried die psigoses in gevalle van verslaafdheid aanhits.[6]

Hierdie toestand, alhoewel dit skrikwekkend is, is goedaardig en 'n 30%-risiko om 'n psigose te ontwikkel, is die moeite werd, indien die uitslag een van lewe of dood is.

1. Van die Redaksie (1956): Brit. Med. J., **2**, 1108.
2. Shaw, F. H., Simon, S. E., Cass, N. M., Shulman, A., Anstee, J. R en Nelson, E R. (1954): Nature, Lond., **174**, 402.
3. Shulman, A., Shaw, F. H., Cass, N. M. en White, H. M. (1955): Brit. Med. J., **1**, 1238.
4. Pedersen, J. (1956): Lancet, **2**, 965.
5. Clemmesen, C. (1956): *Ibid.*, **2**, 966.
6. Kjaer-Larsen, J. (1956): *Ibid.*, **2**, 966.

treatment. Even moderate overdosage with these leads to vomiting, hyperspasticity (even convulsions), hyperthermia and a raised blood-pressure; and in severe cases they have not proved their worth in saving life.

Two years ago Shaw *et al.*[2] reported that a drug they were investigating and which has since been called bemegride (Megimide) possessed 'a pronounced antagonistic effect to barbiturates' in rats with induced acute barbiturate poisoning. They claimed that in these animals the drug shortened the period of coma and restored consciousness more rapidly. Subsequently several successful series of human cases have been reported. Shulman *et al.*[3] found that cases in coma could be brought into a 'safe state'—i.e. a state of consciousness approximating to light anaesthesia—by repeated injections of bemegride intravenously at 5-minute intervals. Although the precise nature of Shulman's 'safe state' has drawn sharp criticism for its vagueness, there is little doubt that bemegride has saved the lives of countless persons who would otherwise have died of acute barbiturate poisoning.

At the same time, the balance of evidence does not support the other claims of Shaw and his colleagues for the drug. Firstly, bemegride does not shorten the period of unconsciousness, although an immediate effect of an injection is to 'rouse' the patient temporarily. This feature was recently corroborated by a Danish clinical team in Copenhagen; within half an hour of an intravenous injection of bemegride, each of 22 deeply comatose patients reacted to strong stimuli and showed reflexes present—'but in most cases the patients lapsed into coma after the first infusion and a further infusion had to be given.'[4] Clearly this property of the drug is potentially life-saving, as Clemmesen (of the same Danish team) showed in 7 cases of respiratory paralysis with total apnoea due to acute barbiturate poisoning. All 7 cases responded briefly but critically to the drug.[5] Shaw's second claim—that bemegride is a direct pharmaceutical antagonist of the barbiturates—appears also to be unsubstantiated. It seems far more likely to be a non-specific central nervous stimulant like picrotoxin, for its administration can result in reflex hyperactivity, electro-encephalographic changes and even psychotic disorders. Of 50 patients in the Copenhagen series, 15 developed a psychotic state characterized by delirium and hallucinations. Nine of these patients were barbiturate addicts, and it is suggested very tentatively that bemegride provoked the psychoses in cases of addiction.[6] This condition, although frightening, is benign and a 30% risk of developing a psychosis is well worth while if the issue is one of life or death.

1. Editorial (1956): Brit. Med. J., **2**, 1108.
2. Shaw, F. H., Simon, S. E., Cass, N. M., Shulman, A., Anstee, J. R. and Nelson, E. R. (1954): Nature, Lond., **174**, 402.
3. Shulman, A., Shaw, F. H., Cass, N. M. and White, H. M. (1955): Brit. Med. J., **1**, 1238.
4. Pedersen, J. (1956): Lancet, **2**, 965.
5. Clemmesen, C. (1956): *Ibid.*, **2**, 966.
6. Kjaer-Larsen, J. (1956): *Ibid.*, **2**, 966.

THE COST OF DIAGNOSIS

Medical progress has laid severe obligations on the doctor. In the matter of diagnosis it is often no longer possible to arrive at a full and complete diagnosis by clinical means alone. A careful history, full clinical examination and long experience will often enable the wise practitioner to arrive at a working diagnosis, and his therapy is based on this working diagnosis. If anything more is required, he has to face the responsibility of advising his patient that ancillary methods of examination are necessary. The cost and number of these have mounted rapidly in the last few years. That this should be so is natural. The examinations of the blood, of the bone marrow and of the various fluids and solids excreted by the patient have increased in number and complexity as our knowledge of disease has increased. The cost also of radiological examinations has kept pace with the rising cost of living and the mounting costs of radiological equipment. These investigations have to be performed by or under the supervision of highly trained medical men, and doctors and technicians have to be adequately paid; the result is reflected in mounting bills. The patient at the end of a disease is often confronted with a surprisingly high doctor's bill for which he has very little to show and he sometimes holds his practitioner to blame for this. The doctor cannot always take the patient fully into his confidence on the question of diagnosis because he must allay fears and not inspire them. His doubts about the diagnosis and the need to exclude various horrible and frightening diseases are something he must keep to himself. The patient only hears about these doubts and difficulties after the diagnosis is settled and he is presented with the bill.

It is a wise policy for the doctor, for his own protection, to take his patient into his confidence as much as he can about the fees that are being incurred, and to remember that in due course there will come a reckoning.

'N PYLVERGIF VAN DIE BOESMANS IN SUID-AFRIKA

U. G. BIJLSMA EN F. DE WAARD

Pharmacologisch Laboratorium, Utrecht, Nederland

Die ondersoek t.o.v. die werking van pylgifstowwe het dikwels in die verlede 'n interessante verloop gehad. Terwyl die ondersoeker hom aanvanklik op suiwer toksikologiese terrein waag, het dit na korter of langer tyd geblyk dat hy eintlik te doen het met stowwe wat van groot betekenis vir die geneeskunde was. As eerste voorbeeld daarvan kan ons curare noem—destyds die berugte gifstof wat die Indiane van die Amazone-bekken aan hulle pylpunte gebruik het, maar tans 'n belangrike farmakon, veral in die chirurgiese en die psigiatriese kliniek. Tweedens kan ouabaïen en strophantien genoem word wat van die belangrikste bestanddele uitgemaak het van die pylgifstowwe wat deur die Oos-Afrikaanse inboorlinge gebruik is, terwyl dit tans, in die moderne geneeskunde, waardevolle terapeutika by die behandeling van hartlyers is.

Die Boesmans van die Kalaharie gebruik o.a. die larwes van 'n kewer *Diamphidia simplex* Péringuey by die vervaardiging van pylgif. Hoewel Duitse ondersoekers hulle reeds besig gehou het met die toksikologie van die gifstof wat uit hierdie larwes verkry is, skyn dit vir ons nuttig om enkele aspekte van die giftige werking van hierdie stof nader te ondersoek. In die besonder het ons onsself afgevra waarom 'n stof wat luidens die literatuur nie 'n spoedige dood van die getroffe dier tot gevolg het nie, nogtans so 'n reputasie as pylgif gekry het.

Een van ons was so gelukkig om tydens 'n besoek aan Suidwes-Afrika 'n 15-tal kokonne van die *Diamphidia simplex* Péringuey in die hande te kry. Maar voordat ons eie ondersoek en bevindinge meegedeel word, gee ons eers 'n kursoriese oorsig oor die oue—hoofsaaklik Duitse—literatuur wat in Suid-Afrika waarskynlik moeilik te raadpleeg val.

In 1894 deel Schinz mee dat hy op 'n terugtog van 'n ekspedisie na die N'gamimeer, 'n aantal larwes van 'n kewer bekom het wat deur die Boesmans by die vervaardiging van hulle pylgif gebruik word. Die ontdekkingsreisigers, Le Vaillant, Livingstone en Baines, het reeds vantevore van hierdie giftige larwes gewag gemaak.

Dit het egter geblyk dat Schinz se eie versameling ongeskik vir entomologiese en farmakologiese ondersoek was. Van 'n vriend het hy gelukkig 'n tweede versameling ontvang wat deur Fairmaire (Parys) en Böhm (Leipzig) ondersoek is. Die Franse insektekundige het 'n latynse beskrywing gegee en die kewers ingedeel as behorende tot die *Chrysomelideae* van die geslag *Diamphidia*, maar noem hulle ten onregte na homself. Prioriteit moet naamlik gegee word aan die naam: *Diamphidia simplex* Péringuey (soms kom 'n mens ook die naam: *Cladocera nigro-ornata* teë). In dieselfde publikasie kon Böhm reeds meld dat die gif 'n toksalbumien is wat taamlik onskadelik vir koubloedige diere maar dodelik vir die soogdiere is. Die fatale uitwerking van die gif word mede bepaal deur die toegediende dosis. Na subkutane inspuiting van 'n waterige oplossing van die inhoud van die larwes, ontstaan hemoglobinurie, diarree en paralitiese verskynsels. By na-doodse ondersoek is ernstige nierbeskadiging, sowel as hemorragiese ontsteking van die dermslymvlies en van die huid rondom die inspuitingsplek vasgestel.

Lewin het in dieselfde jaar sy dissertasie-ondersoek aan die pylgifte gewy. Behalwe die gegewens deur Böhm vermeld, wys hy ook op die intrede van krampe en asemhalingsstoornisse na 'n snelle opname van die gif uit die larwes van *Diamphidia*.

In 1897 het Böhm en Starcke 'n volledige ondersoek gepubliseer. Eersgenoemde het homself hoofsaaklik met 'n ondersoek van die chemiese eienskappe van die

gifstof besig gehou. Maserasie van die larwe in 'n fisiologiese soutoplossing het 'n werksame suspensie gebied (1 larwe per 1 ml.), wat in 'n dosis van 0·5 ml. sonder uitsondering fataal vir konyne was. Nadat die larwes met eter behandel is, is geen giftige stof afgeskei nie en deur dit te kook en met alkohol te behandel, het die giftige stof onaktief geword. Die gif was met ammoniumsulfaat uit te sout, en nie dialiseerbaar nie, met die gevolgtrekking dat die stof 'n toksalbumien is. Die toevoeging van 'n klein hoeveelheid chloroform het die agteruitgang voorkom wat in die werksaamheid intree a.g.v. die ontwikkeling van kokke-kulture. In droë toestand het die gif vir jare lank aktief gebly. Dit het geblyk dat die kokonne en die volwasse kewers nie giftig was nie.

Starcke het die effek van die aktiewe suspensie op verskillende diersoorte ondersoek. Na 'n subkutane toediening aan konyne was die vergiftigingsbeeld as volg: 'n Spoedige gebrek aan eetlus, die dier verloor sy lewenslustigheid en gaan stil in 'n hoekie sit. Na 4–10 uur word bloedrooi urine afgeskei en die faeces word dun. Na ten minste 7 uur tree die dood in met vergiftigingsverskynsels aan die senustelsel wat blyk uit: onmag om alleen op te staan, sensibiliteitsverlies, soms krampe en ten slotte asemstilstand. Met klein dosisse het die diere dae lank aan die lewe gebly—hulle kry later weer eetlus, die urine word weer helder, maar op die plek van die inspuiting ontstaan 'n sweer. Sulke skynbaar geneesde, maar vermaerde diere, gaan tog binne 14 dae dood.

Ook by katte en honde was die verloop van die vergiftiging nie akuut nie. Selfs 6½ uur na 'n binne-aarse inspuiting, was 'n hond nog nie dood nie. By katte is na 1–1½ uur parese van die agterpote opgemerk; ook hier het sensibiliteitsverlies ingetree en met name analgesie. By kronies-verlopende gevalle het subkutane absesse ontwikkel wat perforerende en groot etterrende swere tot gevolg gehad het. In hierdie stadium is 'n temperatuurstyging van 41·3°C gemeet. Mikroskopiese ondersoek van die urine het geen normale eritrosiete, maar wel rooi, silinderagtige vormsels getoon; voorts is eiwit en oksihemoglobien aangetoon.

'n Uitgebreide verslag van die patologies-anatomiese bevindinge, veral t.o.v. die niere is gegee. In laasgenoemde orgaan is veral degeneratiewe veranderinge in bloedkapillêre, glomeruli, tubuli contorti en opstygende been van die Henle se lisse opgemerk.

Plaaslike toediening van die gif op die konjunktiva van konyne het 'n heftige, dog verbygaande, ontstekingsreaksie tot gevolg gehad. Een dier het na ses dae gevrek onder 'n epiloptiform beeld, en 'n ander het na vier maande atrofie van bulbus oculi en nervus opticus vertoon. Anorexie en hemoglobinurie is by hierdie wyse van toediening nie waargeneem nie.

Dit het geblyk dat voëls ook besonder gevoelig vir die gif was na inspuiting daarmee. 'n Duif en 'n mossie is egter glad nie geskaad deur die insluk van een van die larwes nie. Paddas het eers enkele dae na die inspuiting doodgegaan a.g.v. hemorrhagiese veranderinge.

Heubner het van 'n kollega 'n paar vergiftigde pyle uit die gebied tussen Gobabis en Rietfontein, Suidwes-Afrika, ontvang. Twee opmerkinge van die autochtone bevolking word meegedeel: dat die getroffe diere eers na enkele ure doodgaan en dat die jagters vóór die toebereiding van die wildsvleis, die pylpunt met die omringende vleis uitsny.

Hierdie ondersoeker het daarin geslaag om behalwe 'n eiwitagtige gifstof, ook 'n anorganiese vergif te isoleer. Terwyl hy nie van larwes nie, maar van vergiftigde pyle in sy ondersoek gebruik gemaak het, is dit nie bewys dat die anorganiese komponent van *Diamphidia simplex* afkomstig is nie; die Boesmans het immers dikwels mengsels van verskillende gifstowwe, o.a. Euphorbiasappe, *Haemanthus toxicarius* (slanggif) aan hulle pyle gesmeer! Die ontstaan van 'n absederende phlegmoon op die inspuitingsplek by 'n hond wat met gibehandel was, maak die aanwesigheid van die larwegifstof wel waarskynlik.

Hall en Whitehead het gedink dat die Kung- en Heikumstamme ook van bakterieë-vergifte gebruik gemaak het. Hierdie outeurs het ook die oorlewingstye van sommige wildsoorte gemeld, bv. 'n springbok 2 uur, 'n eland 10 uur en 'n kameelperd 50 uur.

In Suidwes-Afrika het Trommsdorf proewe met die larwegifstof gemaak. Die larwes het hy in ruil vir tabak van die Boesmans gekry. Die larwes was, blykens die duidelike afbeeldinge, van dieselfde soort as wat ons ondersoek het. Toksikologies onderskei hierdie ondersoeker 'n plaaslike en 'n algemene werking; die laaste kategorie word deur hom in twee gedeel, nl. verskynsels van neurologiese en van hemorrhagiese ontstekingsaard, waartoe hy ten onregte ook die nierveranderinge reken.

Händel en Gildmeister het kewer-larwes en kokonne uit die gebied van Grootfontein, Suidwes-Afrika, ontvang. Sowel uit lewende as uit dooie materiaal is gif verkry. Hierdie outeurs het die invloed van die gifstof op eritrosiete *in vitro* uitvoerig ondersoek. 'n Suspensie van 1 larwe per 1 ml. kombuissout-oplossing, wat 10,000 maal verdun is, het in 30 min. nog 'n hemoliserende werking op caviabloed. Bloed van voëls en visse is veel minder aangetas. Bloed in isotoniese rietsuikeroplossing was baie weerstandkragtiger teen die hemoliese, behalwe in die geval van visbloed. Leukosiete is deur die gif gedood, maar verskillende soorte bakterieë nie.

Die uitvoering van proewe op lewende diere het weer dieselfde resultate gelewer as die wat deur die reeds genoemde ondersoekers geboekstaaf is. Besonder interessant was die immunisasie-proewe. Deur, met uiters klein dosisse te begin, en die dosisse geleidelik te vergroot, kon konyne ten slotte sonder nadelige gevolge, 200 maal die aanvangdosis, d.w.s. die inhoud van 'n hele larwe, verdra. Die immuun-serum is eers *in vitro* op hemolise-verhindering getoets en daarna *in vivo* toegepas.

'n Ander mededeling van terapeutiese waarde het ons by Waterson gevind. Die Boesmans het nl. 'n natuurlike antidotum, *Kalahuetlwe*, geken—'n bolgewas wat klein, geel blommetjies dra.

EIE PROEWE

Die kokonne is in Desember 1954 deur dr. Watt aan een van ons gegee—'n versameling wat hy in Februarie 1953 van die magistraat van Gobabis ontvang het.

Hulle het gemiddeld ongeveer 500 mg. geweeg en die larwes daarin sowat 150 mg.

'n Giftige suspensie is berei deur 'n snit in die huidjie van die larwe te maak, die jellie-agtige inhoud versigtig uit te druk en dit vir enkele minute te skud met 'n fisiologiese soutoplossing. Die verhouding van 1 larwe per 5 ml. soutoplossing is altyd as 'n standaard suspensie gehandhaaf. Dit was nie moontlik-mede a.g.v. die geringe hoeveelheid materiaal—om betroubare gegewens omtrent die sterkte van hierdie ‚standaard' saam te stel nie, aangesien die larwe-inhoud besonder onvolledig gesuspendeer het.

Tydens oriënterende proewe is 7 uit 9 witmuise gedood en wel binne 'n uur na die inspuiting van 0·1—0·5 ml. van 'n standaard-suspensie, subkutaan sowel as intraperitoneaal. Ons het die indruk gekry dat die gif na enkele ure in werksaamheid verswak het. Die muise het almal verlammingsverskynsels aan die agterpootjies vertoon, terwyl die dood in een geval reeds binne 2 min. na 'n intraperitoneale inspuiting ingetree het, wat gepaard gegaan het met heftige krampaanvalle. Dit is moontlik dat die gif in hierdie geval direk in die bloedstroom ingespuit is. 'n Suspensie van die kokon alleen het ook giftig geblyk te wees—'n feit wat nie met die bevindinge van Boehm ooreenstem nie.

Daar is aan die moontlikheid van 'n curare-agtige komponent gedink. Met 0·1 en 0·3 ml. van 'n standaardsuspensie was daar egter geen curare-werking op twee gedeserebreerde paddas, met afgebinde *A. iliaca* (proef van Claude Bernard), nie. Die paddas was wel die volgende oggend dood. Inspuiting van 0·3 ml. standaardsuspensie in 'n gesonde padda het alleen 'n inisiatiefgebrek tot gevolg gehad. Die gedrag het ooreengekom met dié van 'n gedeserebreerde dier. 'n Werking op die hoëre dele van die sentrale senuweestelsel mag miskien veronderstel word. Origens kon ons 'n mindere gevoeligheid van die padda t.o.v. die gif vasstel. Op 'n geïsoleerde paddahart is geen digitalisagtige werking gekonstateer nie.

Subkutane inspuiting van 0·5 ml. van die standaardsuspensie in 'n kat, het na 20 min. verskynsels laat ontstaan wat aan buikpyn laat dink het: die dier het blykbaar onbehaaglik gevoel, het telkens op 'n ander manier gaan lê, en het af en toe in 'n sywaartse rigting geloop—'n besonder ongewone handelswyse. Na toevoeging van 1·0 ml. het die asemhaling dieper en vinniger geword en daar het geen sianose bestaan nie. Ook die orige 3·5 ml. is toegedien terwyl die gedrag van die kat ewe merkwaardig gebly het; verskeie kere het die kat gemiaau. In die loop van die volgende dag is die dier dood. By seksie was die inspuitingsplek grou van kleur (die onsmaaklikheid, en nie die giftigheid nie, sou die Boesmans daarvan weerhou het om die getroffe liggaamsdeel te eet), hart en longe het geen duidelik waarneembare afwykings getoon nie, aan die niere was veral die donker kleur van die murg opvallend, in die blaas was bloedkleurige urine, aan die maag en derms was geen duidelike afwykinge vas te stel nie, behalwe 'n vaatinjeksie van die serosa. By mikroskopiese ondersoek was daar degeneratiewe veranderinge aan die hart, lewer en niere gekonstateer; in die lewer, gal-thrombi, wysend op verhoogde bloedafbraak; in die niere, tubulusvulling met 'n geel-rooi-bruin inhoud wat deur die glomeruli deurgelaat is—waarskynlik hemoglobien.

Die verskynsels van buikpyn het ons daartoe gebring om 'n kat, na voeding met bariumpap en inspuiting met die giftige suspensie, röntgenologies te ondersoek. By herhaling van die röntgenproewe het geblyk dat die maag veel langsamer ontledig word as wat onder normale omstandighede die geval is, en dat die kolon ook later gevul word. Terwyl in die geval van 'n gesonde kat, na koolhidraat-voeding, die maagontledinging en die begin van die kolonvulling reeds binne 3 uur geskied, was daar by die vergiftigde kat selfs na 6 uur nog geen sprake daarvan nie. Sommige van die X-straalbeelde het die indruk gewek van 'n spasma van die pylorus-deel van die maag—later is dit egter nie meer op die X-straal fotos waargeneem nie. 'n Vertraging van die ontlediging van die maagderm-kanaal is egter by drie katte, uit 'n reeks van ses, met sekerheid vasgestel.

Die katte was gedurende die proewe gedurig onder observasie. Behalwe die verskynsels van onbehae, wat

Fig. 1. Twee katte na inspuiting van die gifstof. Uitgesproke apatie.

ons aan buikpyn toeskryf, het 'n sekere apatie ook opgeval. Die katte het minder gereageer op prikkels en het allerlei klein onaangenaamhede verdra. 'n Voorbeeld van hierdie toestande is duidelik sigbaar op die meegaande foto van twee vergiftigde katte (Fig. 1).

Die vraag het ontstaan waaraan die vertraging van die maagdermontlediging toegeskryf moes word. Daarom is die gifstof ondersoek t.o.v. die werking daarvan op geïsoleerde cavia- en katderms. Dit was duidelik dat die toevoeging van sowel kleinere as grotere dosisse (resp. 0·001–0·01 ml. en 0·125 ml. van 'n standaardoplossing) aan die medium (10 ml. tyrode-oplossing) 'n spasma opgewek het. Die werking van klein dosisse kon nie deur atropien of benodien opgehef word nie, maar wel deur cyclospasmol (amandelsuur ester van 3, 3, 5–trimetiel cyclohexanol), ten minste binne enkele minute na die toediening van die gif. Kort daarna het die dermstukkies so vergiftig geblyk, dat hulle tot geen sametrekking of verslapping gebring kon word nie. (Sien Fig. 2).

Ten einde 'n indruk te kry van die werking van die gif onder natuurlike omstandighede, het ons twee melkbokke daarmee ingespuit en hulle vir enkele ure dopgehou. Vir die kleinste bok (byna 10 kg.) het ons 1·0 ml. van 'n standaardsuspensie onder die rugvel ingespuit. Reeds na 6 minute kon 'n mens merk dat die dier nie lekker voel nie—sy het telkens gaan lê en weer opgestaan.

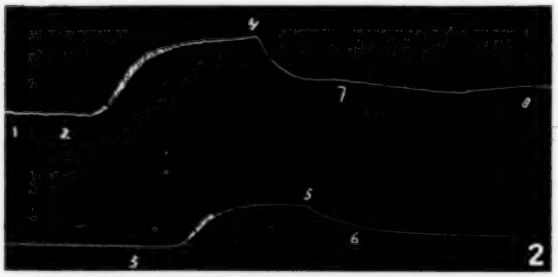

Fig. 2. Werking van die stof op die geïsoleerde katderm en die spasmolitiese werking van cyclospasmol. Beide dermstukkies is in 10 ml. tyrode-oplossing gehang. Standaard-suspensie = 1 larwa gesuspendeer in 5 ml. Ringerse oplossing.
1 = 0·5 ml. van 'n 100-maal verdunde standaard-suspensie.
2 = 0·5 ml. van 'n 10-maal verdunde standaard-suspensie.
3 = 1·0 ml. van 'n 10-maal verdunde standaard-suspensie.
4, 5, 6, 7 en 8 is telkens 0·1 ml. van 'n oplossing van cyclospasmol in propyleenglikol (1 mg./ml.). Omhooggaan van die grafiek beteken sametrekking van die stukkie katderm.

Die bok het op die grond gerol met uitgestrekte pote, het haarself af en toe opgerig, onreëlmatig asemgehaal maar het nie geblêr nie (Fig. 3). Die dier kon wel loop

Fig. 3. Melkbok na inspuiting van die gifstof. Duidelike katatonie.

maar het verkies om in die koelte te bly lê. Die stert is dikwels opgelig in 'n poging om te mis, 'n verskynsel wat op maagpyn wys. As die dier wou opstaan, het sy haarself opgegooi—die bene het blykbaar ondertussen verswak. Die frekwensie van die asemhaling het tydelik tot 180 per min. toegeneem, maar dit het na 2 uur weer tot 60 per min. gedaal. Die mees opvallende verskynsel in hierdie stadium was 'n duidelike apatie, wat soms selfs na 'n katatonie gelyk het. Sekere beweginge het byna die aard van 'n slaapwandelaar geëwenaar. Eetlus het die dier nie getoon nie; sy het haar aan die end van die middag sonder moeite na die stal laat neem, maar die volgende dag was die bok tog dood. By 'n patologies-anatomiese ondersoek is degeneratiewe veranderinge in die niere, hemoglobinurie, en 'n mesenterium met besonder sterk vaatinpomping gekonstateer, maar in die harsingstam geen veranderinge, ook nie mikroskopies nie.

Die groter bok (15 kg.) het vier uur later 'n inspuiting van 3·5 ml. van dieselfde suspensie gekry, maar het veel minder afwykende verskynsels vertoon. Daar is wel tekens van onwelsyn, bv. gebrek aan eetlus en apatie, bespeur. Voorts is ook hier dieselfde moeilikhede as wat by die kleiner bok opgemerk is, gekonstateer, en af en toe het die dier klaend geblêr. Die volgende dag is die dier rustig-weidend aangetref. Na 'n week egter het 'n kwaai sweer van 5 cm. middellyn op die plek van die inspuiting gevorm wat ons genoodsaak het om die dier dood te maak.

Wat is nou die betekenis van hierdie vergif van die Boesmans? Ons dink dat dit in die eerste plek 'n immobiliserende uitwerking het. Van die tallose diersoorte wat ondersoek was, het dit volgens die literatuur en ons eie waarneminge geblyk dat geen enkel een direk gedood is nie. (Ons moet wel daarop wys dat by die toksikologiese ondersoeke nie so swaar gedoseer is soos die Boesmans dit gewoonlik gedoen het met 'n pasta wat uit verskeie larwes berei is nie). Daar tree in elk geval baie duidelike tekens van onwelsyn in en daarna 'n toestand van apatie, wat die wild sou noop om die vlug te staak, sodat die gif sy dodelike werking in die betreklike nabyheid van die jagters kan voltrek.

In hoeverre die onbehaaglike gevoel deur die aangetoonde werking op die maagdermkanaal veroorsaak word, kan nie met sekerheid gesê word nie, maar die apatie moet aan 'n werking op die sentrale senuweestelsel toegeskryf word. Daar is wel een proef gedoen t.o.v. die invloed op die kniebuigings-refleks (N. peronaeus) van 'n gedekapiteerde kat. Baie klein dosisse (1–3 ml. van 'n 200 = maal verdunde standaard-suspensie) intraveneus toegedien, het die refleks verhoog.

Vir die toesending van nuwe gifstof hou ons onsself aanbevole.

Graag betuig ons dank aan dr. J. S. Watt, Direkteur van Landbou. S.W.A. Administrasie, Windhoek, vir die verstrekking van die kokonne van *Diamphidia simplex* en aan die navorsingsafdeling van die N.V. Brocades en Stheeman vir die cyclospasmol.

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ADDENDUM

A BUSHMAN ARROW POISON

(*Diamphidia Nigro-ornata Stal.*)

DOUW G. STEYN

Professor of Pharmacology, University of Pretoria, Pretoria

Some 3½ years ago, through the kind services of Dr. J. S. Watt, Director of Agriculture, South West Africa Administration, Windhoek, S.W.A., I obtained 20 cocoons of the beetle *Diamphidia nigro-ornata Stal* (*D. simplex* Pèringuey) from the Epicuro Reserve, situated North-East of Gobabis. The cocoons are found in the sandy soil under small shrubby bushes. The shells are hard and dark brown in colour, and are composed of fairly coarse sand-granules glued together. The inside of the cocoon shell is black. Each cocoon contains a single larva which lies in a doubled-up position and is light orange-pink in colour. (See figs 4-7). The wieghts were as follows: 20 cocoons (unopened) 9·4 g., 20 cocoon shells 6·4 g., 20 larvae 3·0 g., 11 semi-dry larvae 0·7 g.

EXPERIMENTS ON RABBITS

The experiments detailed below were conducted 3 years ago. The material used for injection into rabbits (cocoon shells or dry larvae or fresh live larvae) was finely ground up in a mortar and, after the addition of physiological saline solution, further ground until an extremely fine suspension was obtained. This was filtered through very ane cheese-cloth before being injected.

The result of subcutaneous, intramuscular and intravenous injections are shown in Table I.

DISCUSSION

In the course of investigations in the Northern parts of South West Africa, I was informed by various individuals that the Bushmen do not use the above larvae in shooting game but reserve them for protection against their enemies, because they (the larvae) immobilize the victim immediately and cause death within a few minutes. These two facts appear to be borne out by the results of these experiments. Slower types of

TABLE I. THE EFFECT OF THE COCOONS AND LARVAE OF DIAMPHIDA NIGRO-ORNATA STAL. ON RABBITS

Rabbit No. and Weight	*Material*	*Volume Injected (c.c.)*	*Equivalent*	*Result*
A 2·0	Dry cocoon shells	2·5 sc	1·5 g. dry cocoon shell	Negative.
B 2·0	ditto	2·5 IM	1·5 g. ditto	Negative.
C 2·3	ditto	2·5 IV (Left ear vein)	1·5 g. ditto	Almost immediately developed miosis, restlessness, accelerated pulse, dyspnoea, paresis and paralysis. Frequent urination and defaecation. Urine reddish (haemoglobinuria). Died within 6 hours after injection. No autopsy.
D 2·5	Dry larvae (as submitted)	2·5 sc	0·175 dry larvae	Miosis within 2 minutes (further than C). Died within 6 hours after injection. Urine reddish. No autopsy.
E 2·6	ditto	2·5 IM *	0·175 g. ditto	As in C and D. Died 4 hours after injection. Injected hind leg completely paralysed within ½ hour. Urine reddish. No autopsy.
F 3·5	ditto	0·5 IV	0·035 g. ditto	Immediately after injection very restless, staggering, severe general convulsions, gasping for breath and died ¾ minute after injection. Autopsied immediately: Blood in right atrium and ventricle completely coagulated and partly coagulated in left atrium and ventricle and the coronary arteries.
G 2·1	Fresh live larvae	0·5 sc	0·075 g. fresh larvae	Miosis, dyspnoea, restlessness, and accelerated pulse within 10 minutes after injection; recovered within an hour.
H 1·8	ditto	2·5 sc	0·375 g. ditto	Fatal convulsions within ½ minute after injection. Autopsy immediately: Similar to that of F.
I 2·5	ditto	0·025 IV	0·25 mg. ditto	Developed no symptoms.
J 1·9	ditto	0·1 IV	1·0 mg. ditto	Within 30 seconds miosis, restlessness, dyspnoea, accelerated pulse, generalized tremors followed by convulsions, and death within 2 minutes after injection. Autopsy as in F.
K 2·3	ditto	0·1 IV	1·0 mg. ditto	Generalized convulsions and death while the injection was was being made. Immediate autopsy: blood in both atria and ventricles and coronary vessels completely coagulated.
L 1·8	ditto	0·25 IV	2·5 mg. ditto	As in K; died within 1½ minutes after injection. Autopsy: As in K; also haemolysis.
M 3·2	ditto	0·5 IV	5·0 mg. ditto	Restlessness and miosis in the course of the injection, and death in generalized convulsions, as in the other cases, within ¾ minute after injection. Autopsy: As in K.
N 1·7	ditto	1·0 IV	10·0 mg. ditto	Death in generalized convulsions within 1 minute after injection. Autopsy: As in K; also pronounced haemolysis.

sc = subcutaneously. IM = intramuscularly. IV = intravenously into vein of left ear. * into muscle of left hind leg.

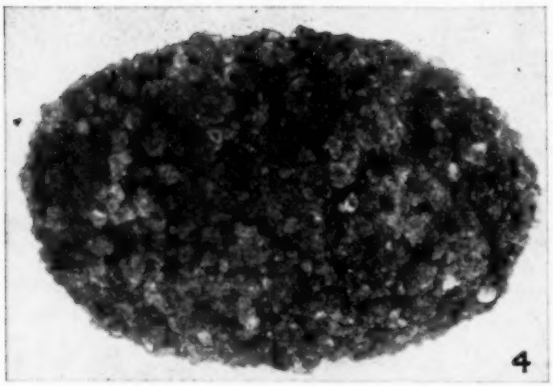

Fig. 4. Cocoon of *Diamphidia nigro-ornata Stal.* Natural size $\pm\frac{3}{4}-\frac{1}{2}$ by $\frac{1}{4}-\frac{1}{2}$ inches.

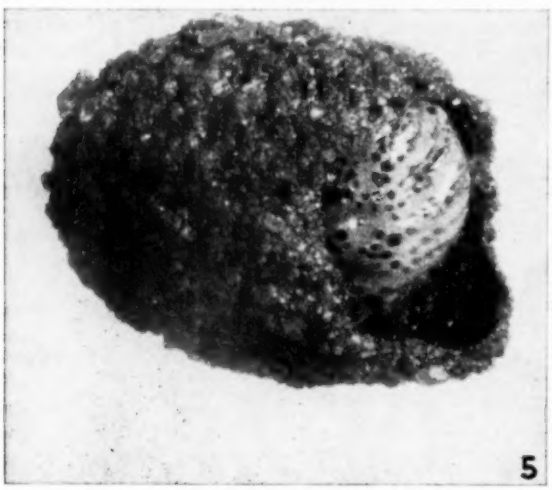

Fig. 5. Cocoon of *Diamphidia nigro-ornata Stal* with one end opened to expose the larva.

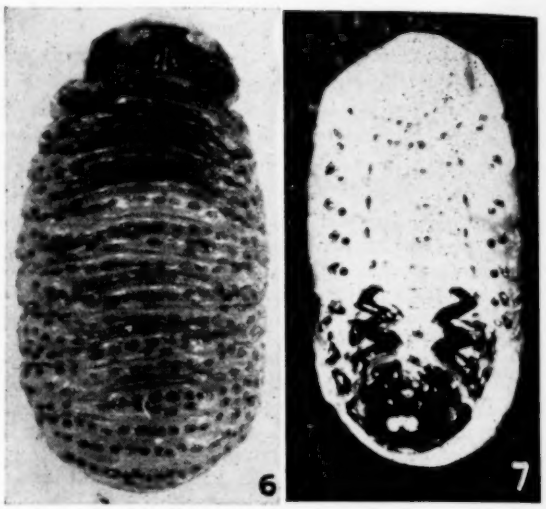

Fig. 6. Larva of *Diamphidia nigro-ornata Stal* removed from the cocoon. Natural size $\pm\frac{3}{4}-1$ by $\frac{1}{4}-\frac{1}{2}$ inches.

Fig. 7. Cocoon of *Diamphidia nigro-ornata Stal* showing position of larva in cocoon.

poison (*Adenium boehmianum*, etc.), are used for game hunting.

From the foregoing experiments it appears (1) that the cocoon shell also contains a small amount of the poison present in the larva, and (2) that the dried larvae submitted retained their toxicity. The quantity of material available was insufficient to ascertain whether in drying they had possibly decreased in toxicity.

As was to be expected, the poison is most toxic when injected intravenously. According to these preliminary tests the intravenous minimum lethal dose for the rabbit appears to be between 0·25 and 0·5 mg. of fresh larva per kg. body-weight.

THE TREATMENT OF CARCINOMA OF THE CORPUS UTERI*

BRYAN C. MURLESS, B.A., M.B., B.CH. (CANTAB.), F.R.C.S.E., F.R.C.O.G.

Senior Obstetrician and Gynaecologist, King Edward VIII Hospital, Durban, and Lecturer to Department of Obstetrics and Gynaecology, Natal University. Assistant Gynaecologist, Addington Hospital, Durban

In recent years carcinoma of the cervix has been much to the fore but cancer of the body, 'the poor relation', has received less attention. In most cases cancer of the body is treated by general practitioners, general surgeons and most gynaecologists throughout the country by total hysterectomy and removal of the adnexa. This is not surprising, for this treatment has been the text-book teaching almost without change since 1905, except for the addition of radiotherapy by some surgeons in the last decade. It is the object of this paper to argue that this treatment is insufficient and that, with present-day conditions, a more radical approach is justified.

* Paper presented at the Congress of the South African Society of Obstetricians and Gynaecologists, Durban, July 1956.

Methods of Treatment

Several variations in the treatment of cancer of the body may be listed:

1. Surgery alone (total hysterectomy and removal of the adnexa).

2. Radiation alone.
3. Surgery + post-operative radiation.
4. Surgery + pre-operative radiation.
5. Vaginal hysterectomy.

Each of these methods has certain shortcomings to which I shall refer below.

RESULTS OF TREATMENT

Truly comparative results are very difficult to obtain for the following several reasons:

1. The variations in treatment listed above.
2. Reluctance of surgeons to publish *absolute* survival rates.
3. The lack of a satisfactory method of clinical 'stageing' of the cases.
4. The small prognostic value of pathological grading owing to the fact that the gross pathological type of growth seems to have no bearing on the recurrence rate (Rickford, 1954).

Broadly speaking, absolute 5-year survival rates reported by well-known clinics for cancer of the body are in the region of 55-60%. It is possible that even these figures are too generous. They mean that 40-45 patients out of every 100 seen die within 5 years of treatment. Also it must be remembered that cancer of the body is often a slow-growing disease, especially when it occurs in old people, and that treatment may merely retard the growth; hence a 5-year survival should not in all cases be considered a cure, for in the Stockholm series (Heyman, 1954) almost 20% of 5-year survivors died of cancer before the 10th year. Most other 10-year published results are equally disappointing. Figures of between 70 and 80% 5-year survivals appear in the literature from time to time, but these are from selected operable cases with the growth confined to the uterine cavity.

The question remains: Are these results acceptable in a disease which is known to be of comparatively low malignancy? My submission is that they are not, and that a certain amount of dangerous complacency exists in our management of this condition. How can these figures be improved?

RECURRENCES

A study of recurrences in cases of carcinoma of the corpus uteri shows that they occur at characteristic sites. Following surgery, a recurrence may be found (1) *In the vagina*, (2) *as a pelvic mass*, *or* (3) *in the pelvic lymph-nodes*.

1. *Vaginal Recurrences*

Vaginal recurrences are found (*a*) in or around the scar at the vaginal vault, or (*b*) low down in the vaginal wall, 1 cm. from the urethral meatus.

(*a*) *The vaginal vault* is by far the commonest site for recurrences in cancer of the corpus. They may be due either to implantation at the time of operation, or lymphatic spread. The weight of evidence is in favour of implantation. Firstly, it has been shown that these recurrences can be avoided with adequate precautions, viz. cervical occlusion and radiation of the vault. Secondly, similar recurrences were common in the suture line after the operation of restorative excision of the rectum for cancer, until it was found that they could be avoided by clamping and thoroughly irrigating the bowel below the growth with perchloride of mercury solution. Thirdly, there is no anatomical reason why lymphatic spread should lead so frequently to the vaginal vault.

(*b*) The para-urethral recurrence is less common, but Meigs (1929) reported them in 12% of cases. They are almost certainly of embolic origin. Evidence in support of this statement is as follows:

(i) Similar recurrences occur in cases of hypernephroma and it is generally conceded this growth spreads chiefly by the blood stream.

(ii) Anatomically, their position coincides with the area of anastomosis between the pudendal arteries and the descending branches of the utero-vaginal vessels from above, a likely place for emboli to settle.

(iii) Experience shows that these recurrences do not appear at long intervals after removal of the primary growth, again suggesting their embolic origin.

(iv) They are often associated with secondary deposits in the lungs (Way, 1951). On the other hand, a few cases surviving a number of years after treatment of these secondaries have been reported. Radiotherapy must therefore be persisted with in all cases.

2. *Recurrence as a Pelvic Mass*

The recurrence which appears as a pelvic mass is characteristic. Clinically it is moderately mobile and feels unattached to the pelvic wall. Therefore it is easily distinguished from recurrence in the pelvic lymph-nodes. For convenience I shall refer to it as the 'unattached' recurrence. It may occur from (*a*) lymphatic spread to the broad ligament or (*b*) intra-abdominal spill. The former explanation seems more likely, although spill cannot be ruled out, for single secondaries have been found on the small intestine and in the abdominal scar.

3. *Recurrence in Pelvic Lymph-nodes*

Lymph-node recurrence from cancer of the fundus has been considered uncommon. However, the following figures are available concerning its incidence:

Brunschwig and Murphy (1954): 17% involved in 57 cases and 23% in 17 late cases requiring partial exenteration.

Javert (1952): 28% in 50 cases (no grade stated).

Winterton (1954): 7·3% in 28 cases.

Kimbell (1956): 20% have nodes involved.

Therefore it seems that lymph-node involvement in this disease is indeed a factor worthy of consideration and must influence our scheme of treatment.

Doubt still exists about the exact anatomy of the lymphatics draining the fundus. For convenience the space between the top of the cavity and the internal os may be divided into 3 equal parts (here named sections 1, 2 and 3—see Fig. 1). The drainage from each section may differ considerably and influence treatment. Two main routes exist:

(i) *The Ovarian Route:* Lymphatics following the ovarian blood supply and draining to the aortic nodes.

(ii) *The Cervical Route:* Lymphatics draining *via*

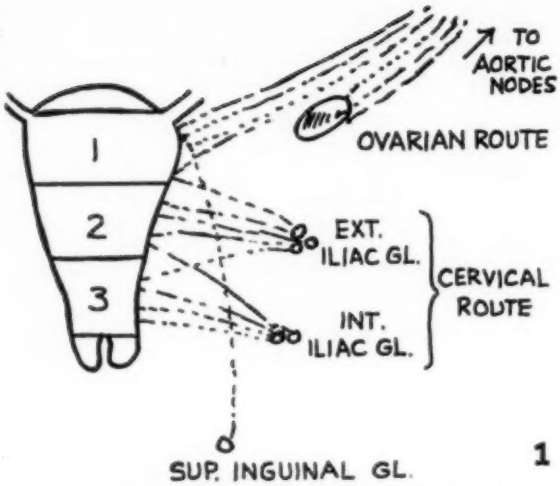

Fig. 1. Schematic drawing of probable lymphatic drainage of corpus uteri.

the parametrium to the same lymph-nodes as drain the cervix.

In all probability section 1 drains mainly by the ovarian route and, fortunately, experience has shown that metastases from this section are rare. That this section can occasionally drain by the cervical route has been demonstrated by Kottmeier (1954) who, injecting radio-active gold beneath the endometrium at the fundus, was able to demonstrate it in the iliac and hypogastric nodes.

Moreover, Várady (1947), carrying out a hysterogram, inadvertantly demonstrated lymphatic emboli travelling to the internal iliac nodes, which they reached in 50 minutes.

Sections 2 and 3 probably drain by the cervical route to the iliac and hypogastric glands. If it can be presumed that the general scheme of lymphatic drainage follows the blood vessels, this reasoning is developmentally sound, for in foetal life the long Müllerian duct is supplied by the uterine artery and it is only later in development that the ovario-uterine anastomosis is formed to supply that part of the duct which is to become the tube.

METHODS OF TREATMENT

Position of Growth

The importance of the position of the growth as regards treatment is therefore obvious. There are 3 ways of ascertaining the site: (1) Hysterography, (2) fractional curettage, and (3) inspection of the cut uterus after hysterectomy.

Hysterography for this purpose is inaccurate. A very small growth may not show at all and the degree of downward extension, which is of vital importance, is always doubtful. Moreover, it would seem there is a real danger of pushing cancer cells into the abdomen. These cells have been demonstrated in the tubes after hysterography and Willis (1948) has described a direct implant on the ovary.

Fractional curettage is also inaccurate for the purpose. More often than not, semi-solid growth flows out as soon as the cervix is dilated. If the tumour is polypoid, the site of origin cannot always be demonstrated with the curette, and distortion by fibroids may further complicate the situation. However, it must be emphasized that the curette remains a valuable instrument for demonstrating or excluding an endocervical growth.

By examination of the cut uterus, the degree of spread can be judged accurately enough with the naked eye and the case labelled according to the section involved. The position of the growth might well be utilized as an accurate method of post-operative 'stageing' e.g.; Stage I, confined to section 1; stage II, involving sections 2 and 3; stage III, direct spread beyond the uterus or the pelvic lymph-nodes involved. A follow-up of each group would produce valuable information.

Shortcomings of Treatment

All methods of treatment in use at present are open to criticism.

Pan-hysterectomy alone fails to guard against parametrial and nymph-node recurrence from a growth that has invaded sections 2 and 3 of the cavity. Even with adequate cervical occlusion, it is not certain that secondary deposits in the vaginal vault will be prevented.

Radiation alone produces good results in very expert hands, as shown by Heyman *et al.* (1941) in Stockholm and Hurdon (1942) in Britain. But radiation alone as a cure for cancer of the fundus remains a blind 'hit-or-miss' type of therapy. Accurate irradiation of a growth is difficult when the extent of spread and depth of myometrial penetration are unknown, when the bulk of the tumour in the cavity varies, and when the cavity itself may be distorted and enlarged by fibroids—commonly associated with carcinoma of the body. Added to this, the permanent response of adenocarcinoma to radiation, generally speaking, is less favourable than that of the squamous variety. Moreover, radium treatment is not without danger. The packing of multiple radium-containers into a thinned-out uterus can easily cause rupture, and it is significant that in the Newcastle series (Way 1951) the operative mortality of radium treatment was 6%—1% more than for total hysterectomy. Ingenious methods have been devised by Green (1954), who uses a balloon containing radio-active tantalum, and Strickland (1953), who uses an intra-uterine spring applicator containing radio-active cobalt. Results of these methods are awaited but the objections mentioned apply in principle.

Surgery + post-operative radiation is still used by some surgeons. Its only object can be to avoid recurrence in the vaginal vault, and Dobbie (1953) has shown that this can be achieved with comparatively small dosage. If this is the object in view, it is difficult to see any advantage over pre-operative radiation, where it seems the distribution should be more effective, and damage to the bladder and rectum less likely. The chief use for post-operative radiation is to salvage the surgeon's mistakes where a surprise carcinoma is discovered after an incomplete operation.

Surgery + pre-operative radiation can be used (*a*) with the object of destroying the growth before surgery,

(*b*) to prevent vault recurrence, (*c*) to treat vault recurrence already present if one believes it can arise from lymphatic spread, (*d*) to 'firm up' the uterus before operation, or (*e*) to reduce sepsis before operation. This method of treatment fulfils many of our requirements, but it still fails to guard against the 'unattached' parametrial recurrence and recurrence in the pelvic lymph-nodes.

Vaginal hysterectomy, as practised by Bastiaanse (1952) in Amsterdam, carries a very low operative mortality rate and high operability rate, but Bastiaanse admits that in 28·7% of his cases one or both ovaries could not be removed. Having recently dealt with 2 cases in which an ovary was involved by growth, we prefer the abdominal route.

Treatment Suggested

What then is the correct treatment for cancer of the fundus uteri? If it can be shown that more radical surgery does not materially increase the operative risk, extension of the operation is justified.

For the last 6 years I have been treating this condition by Wertheim's hysterectomy, with the addition, in the last 4 years, of pre-operative radiation.

Radium is used with the object of avoiding recurrence in the vaginal vault, and a single Stockholm application—50 mg. in cavity and 30 mg. in each fornix, for 24 hours—is applied, if possible immediately after curetting. Using this procedure since 1947, Arthure (1956) says he has seen no vaginal recurrence. There is no object in traumatizing the tissues with full dosage if radical surgery is to follow. Operation takes place 2 weeks after radium application. The cervix is occluded by suture after plugging with ribbon gauze soaked in iodine.

Percival (1952) has described a special clamp for this purpose but suture and plugging should be safe if pre-operative radium is used.

The uterus is removed by Wertheim's method, the upper third of the vagina being removed with the uterus. The uterus is then opened and inspected. The lymph-nodes are removed in all cases except where a small growth is confined to section 1 in old patients whose condition suggests that this additional trauma might be dangerous.

My reasons for recommending this method of treatment are as follows:

1. It is a threefold guard against vault recurrence, embodying as it does (*a*) cervical suture, (*b*) pre-operative radiation and (*c*) removal of the upper third of the vagina.

2. It is a guard against the 'unattached' recurrence by wide removal of the parametrium.

3. It is a guard against lymph-node metastases. It has always been a sound principle of cancer surgery to remove the lymph-nodes draining the affected area. Why should cancer of the body be the sole exception?

4. Since there is no accurate method of defining the extent of spread in the uterine cavity, radical surgery is indicated in all cases. Granjon *et al.* (1954), Swinton *et al.* (1954), Meigs (1955), Brunschwig and Murphy (1954), and Winterton (1954) have expressed similar views.

Results

Five-year figures for radical surgery are few and far between, and this is to be expected because most who favour this treatment have only recent cases to report. My own series is too small to be significant. Winterton (1954), however, had 61 5-year cases, which had received no pre- or post-operative radiation, and which obtained an absolute 5-year survival rate of 76%, or 82% if calculated on the cases in which macroscopic removal was complete. Only one case seen during this time was refused operation. This shows what may be achieved by radical surgery alone and opens up the possibility of even better results with the addition of pre-operative radiation.

Risks of Treatment

It has always been argued that patients with carcinoma of the body of the uterus and its associated disturbances such as hyperpiesis, obesity and diabetes are, generally speaking, not good surgical risks. But it must be remembered that, if this is so, such patients are poor risks for simple total hysterectomy and even for radiotherapy alone. However, experience has shown that, with modern anaesthetics and post-operative intravenous therapy, these old ladies stand up to the operation surprisingly well and that, if any extra risk can be attributed to more radical surgery, it is minimal and far outweighed by the advantages obtained.

Today, age should rarely be a contra-indication to operation and no case should be refused on the grounds of obesity, for adequate exposure can always be obtained with the transverse incision. Hence operability rates are usually high. Winterton (1954), in 90 cases, reports an operability rate of 95%, all radical surgery; Rickford (1954), in 174 cases, 87·4% (some radical surgery); Hawksworth (1954), in 184 cases, 89% (all panhysterectomy); and Kimbell (1954), in 245 cases, 90% (all panhysterectomy).

From figures obtainable, it would seem that the operative risk of radical surgery is no greater than that for simple panhysterectomy in these cases.

Pulmonary embolism still remains the greatest hazard, but its incidence is not increased by the more extensive operation. For radical surgery, Winterton (1954) reports a mortality rate of only 3·5% in 90 cases, while Brunschwig (1954) had no death in 57 cases. For simple panhysterectomy for corpus cancer, Hawksworth's mortality rate (1954) was 4·9%, while Kimbell (1954), in 221 collected cases, quotes a figure of 2·7%.

SUMMARY

1. The treatment of cancer of the corpus uteri has made comparatively little advance in the last 50 years and we have remained satisfied with poor results in a disease where spread is neither early nor rapid. Methods of treatment in use at present are listed and criticized.

2. The recurrences in this disease occur at characteristic sites. The possible causes of these recurrences are studied and the importance of lymph-node metastases emphasized.

3. The position of the growth in the uterine cavity

is of first importance and must influence the treatment required to eradicate the disease.

4. A combination of radical surgery and pre-operative radiation is advocated as a routine treatment in most cases of cancer of the corpus. The reasons for advising such treatment are summarized.

5. The writer submits that the time has come to 'unteach' the established dogma that total hysterectomy with removal of the tubes and ovaries is the treatment of choice in cancer of the body, and that our students should now be taught that a more radical approach is necessary in this dangerous disease.

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THE EFFECT OF VITAMIN C UPON THE VOLUME OF GASTRIC JUICE AND ITS HYDROCHLORIC-ACID CONTENT*

B. J. MEYER, D.Sc. M.B., Ch.B

Department of Physiology, University of Pretoria

Z. M. SMIT, M.Sc.

National Nutrition Research Institute, South African Council for Scientific and Industrial Research, Pretoria

A. C. VOS, D.Sc.

Department of Physiology, University of Pretoria

Our knowledge of the production of hydrochloric acid by the stomach is still very incomplete. There is, however, general agreement that the acid is secreted by the oxyntic cells of the fundus glands, and that both components of the acid, viz. hydrogen and chloride ions, come from the blood. How these cells are able to secrete an acid whose hydrogen-ion concentration is some three million times as high as that of the blood remains at present a mystery. Of the different theories proposed to explain this process, the electro-osmotic theory of Hollander[1,2] and the carbonic acid anhydrase theory of Davenport and Fisher[3] attracted much attention. The latter theory is based on the fact that carbonic acid anhydrase is present in the stomach, and, moreover, in the oxyntic cells only.

Although the exact mechanism whereby hydrochloric acid is produced is not known, different physiological mechanisms and chemotherapeutic agents are known to exert an influence on its production.

All digestive stimuli, of whatever nature, physical or physiological, motor or sensory, visual, olfactory or gustatory, eventually reach the cerebral cortex, where responses are relayed to the thalamus and hypothalamus. From the latter, impulses are transmitted along the vagus and sympathetic nerves.

The sympathetic nerves stimulate the outpouring of adrenaline, which mobilizes the sugar in the bloodstream.

In addition to the neurogenic stimuli arising in the autonomic centres in the hypothalamus, Hume[4] produced evidence that a hypothalamic hormone is produced which exerts a stimulating effect on the anterior pituitary, as a result of which adrenocorticotropic hormone (ACTH) is released, which stimulates the secretion of corticoids by the adrenal cortex. There are also indications that adrenaline stimulates the secretion of ACTH, and thus indirectly the secretion of corticoids.[4,5] If this is so, it becomes a matter of interest that a reaction starting in the suprarenal medulla should induce a defence response through devious channels at the level of the adrenal cortex, the ultimate goal of which is the prevention of cell break-down and histamine release.[6-8]

As far back as 1936, Selye[9] found that gastro-intestinal ulcers and other manifestations of stress were severer in adrenalectomized than in intact animals, and could be diminished by treatment with cortical extracts. Various investigators demonstrated an increase in the gastric secretion of hydrochloric acid and pepsin following the administration of ACTH.[10-13] With Addision's

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disease, gastric secretion is depressed[14,15] and treatment with cortisone may restore it.[16,17] Conversely, the administration of cortisone increases the acidity of the gastric juice,[18] and may aggravate or even produce peptic ulceration.[19]

It is generally believed that a close relationship exists between vitamin C and adrenocortical function. If one accepts an intimate relationship between the adrenal cortex and gastric secretion, a similar relationship might be expected to exist between vitamin C and gastric secretion. In this respect the experiments of Ungar[6] are of interest. He produced numerous gastro-intestinal ulcers and haemorrhages and even perforation by means of severe traumata. These ulcers and other lesions could be prevented by high doses of vitamin C. Similarly, McKee *et al.*[20] showed that vitamin C plays a most important role in the hormonal reaction to stress.

Some surgeons prescribe high doses of vitamin C after a subtotal gastrectomy.[21] This treatment is based on the assumption that vitamin C stimulates the formation of scar tissue and hence the healing of wounds.

Assuming the apparent relationship between the adrenal cortex and gastric secretion, and between vitamin C and the adrenal cortex, as well as the possibility that vitamin C might be of importance in the treatment of peptic ulceration and other gastric disorders, it was decided to investigate the effects of different doses of this vitamin on the volume of gastric juice and the amount of hydrochloric acid secreted.

EXPERIMENTS

A group of 12 volunteers, males and females from the second and third year medical classes were used in this experiment. Their ages varied between 19 and 26 years, with a mean age of 22 years. None of the subjects gave a history of peptic ulcer or antacid therapy. Each subject served as his own control, and was instructed to take no food from 10 p.m. on the evening previous to the experiment until after the termination of the experiment on the following day.

A Ryle's tube was passed into the stomach at 7.50 a.m. on the following day, and all the gastric juice sucked off by means of a 20-c.c. syringe connected to the tube and with the subject alternatively in the erect, right lateral, left lateral, anterior supine and posterior supine positions.

The Ryle's tube was then connected to a suction pump and continuous suction applied from 8 a.m. until 9 a.m. The volume of gastric juice sucked off was read every 15 minutes. At 9 a.m. the tube was disconnected, 30 c.c. of water was taken and suction was suspended until 10 a.m., when all the gastric juice was once again sucked off with the subject in the 5 positions mentioned above. Once more the Ryle's tube was connected to the suction pump and continuous suction carried out from 10 to 11 a.m. in the same way as during the first hour. The experiment was repeated 3 days later but on this occasion the subject was given orally at 9 a.m. 500 mg. of vitamin C, dissolved in 30 c.c. of water. (See Table I).

Four days later the whole experiment was repeated on the same subject, but on this occasion suction was suspended for 2 hours instead of 1 hour between 9 and 11 a.m., and suction was continued at 11 a.m. and maintained until 12 noon., when the experiment was terminated. Three days later the latter experiment was repeated but on this occasion the subject was given orally 1,000 mg. of vitamin C, dissolved in 30 c.c. of water at 9 a.m. (See Table II.)

The visible presence of bile in the gastric juice was regarded as a definite sign of regurgitation of intestinal contents. In cases where this occurred, the experiment was terminated and repeated 3 days later.

The volumes of gastric juice were measured, and the free as well as the total acid was determined by titration against a standard solution of sodium hydroxide with phenolphthalein and p-dimethylaminoazobenzene as indicators. The hydrochloric acid values were calculated in clinical units, mg.-equivalents per litre and milli-equivalents.

Complete 24-hour urine specimens were collected, starting at 8 a.m. on the morning preceding the experiment and terminating at 8 a.m. on the morning of the experiment. A second 24-hour urine specimen was collected on the day of the experiment. In a number of cases a third 24-hour urine specimen was collected on the day after the consumption of 1,000 mg. of vitamin C.

The urine specimens were analysed for total neutral 17-ketosteroids and 17-ketogenic steroids. The 17-ketosteroids were extracted according to the method described by Drekter *et al.*[22] The technique of Norymberski *et al.*[23,24] was adapted for the extraction of the 17-ketogenic steroids. Holtorff and Koch's method[25] was employed for the colour development of both fractions. The latter method was improved by extracting the colour after it had developed, with a mixture of ether and 60% alcohol according to the principles outlined by Zimmermann *et al.*[26] The typical colour is retained in the organic phase, while the contaminent yellow-brown colour remains in the aqueous phase.

RESULTS

The volumes of gastric juice in millilitres and the free hydrochloric acid in milli-equivalents, for both control and experimental periods are tabulated in Table I and II.

Altogether 30 control 24-hour urine specimens from males and 19 from females were analysed. Males excreted an average of 18·3 mg. of neutral 17-ketosteroid per day. All the values were between the extremes of 7·7 and 35·1 mg. per day. The average 17-ketogenic excretion was 12·3 mg. per 24 hours with a range of from 6·1 to 20·2 mg. The average for females were 9·8 mg. of neutral 17-ketosteroids and 8·9 mg. of 17-ketogenic steroids per 24 hours with extremes of 4·8 and 20·3 mg., and 2·6 and 19·7 mg., respectively.

The results were subjected to a critical statistical analysis, mainly by using non-parametric methods—the sign test of Fisher,[27] Wilcoxon's two-sample test, [28,29] Hemelrijk's test of symmetry, [30,31] Kendall's method of M-rankings,[32] and Terpstra's T-test against trend.[33]

When examined at the 5% level the statistical analysis showed that a significantly greater volume of gastric juice was secreted during the first control experiment.

TABLE I. THE VOLUME AND FREE-HYDROCHLORIC-ACID CONTENT OF THE GASTRIC JUICE OF HEALTHY VOLUNTEER SUBJECTS UNDER CONTROL CONDITIONS AND AFTER THE INGESTION OF 500 MG. OF ASCORBIC ACID

Subject No.	Control Experiment				500 mg. Ascorbic Acid			
	8-9 a.m.		10-11 a.m.		8-9 a.m.		10-11 a.m.	
	Volume ml.	Free HCl mEq.	Volume ml.	Free HCl mEq.	Volume ml.	Free HCl mEq.	Volume ml.	Free HCl mEq.
1	57	1·75	41	1·55	31	0·45	37·5	1·34
2	22	0·90	19·5	0·70	23	0·41	27	0·93
3	89	4·32	38	1·39	38	1·87	14	0·66
4	84	0·75	68	2·32	38	0·63	42	1·21
5	103	8·14	95	7·21	61	5·53	63	4·96
6	58	2·03	66	1·05	58	1·85	46	1·47
7	75	2·33	90	1·42	65	1·46	61	1·40
8	140	12·74	104	8·44	98	8·63	55	5·64
9	37	1·95	38	0·90	32	1·60	40	2·28
10	149	6·47	106	6·63	90	6·70	140	7·84
11	22	0·29	16	0·47	21·5	0·44	27	0·67
12	79	2·95	57·5	1·01	67	1·79	64	1·02

TABLE II. THE VOLUME AND FREE-HYDROCHLORIC-ACID CONTENT OF THE GASTRIC JUICE OF HEALTHY VOLUNTEER SUBJECTS UNDER CONTROL CONDITIONS AND AFTER THE INGESTION OF 1,000 MG. OF ASCORBIC ACID

Subject No.	Control Experiment				1,000 mg. Ascorbic Acid			
	8-9 a.m.		11-12 a.m.		8-9 a.m.		11-12 a.m.	
	Volume ml.	Free HCl mEq.	Volume ml.	Free HCl mEq.	Volume ml.	Free HCl mEq.	Volume ml.	Free HCl mEq.
1	32	0·64	40	0·72	30	1·23	30	0·62
2	28	0·0	19	0·17	28	0·92	16	0·03
3	39	2·46	30	1·47	21	1·15	24	1·21
4	40	0·78	35	0·68	50	1·15	50	1·42
5	37	2·70	86	6·06	43	2·04	40	1·76
6	48	2·25	36	0·70	50	0·0	26	0·34
7	52	1·41	41	0·49	75	3·82	64	2·11
8	67	2·38	83	4·56	50	2·00	50	1·70
9	16	0·74	15	0·45	13	0·22	20	0·72
10	62	0·25	60	1·32	70	3·71	76	4·25

This was apparently due to psychic stimulation caused by the strangeness of the situation and the variable degree of difficulty encountered during the initial introduction of the tube. The effect of these stimuli wore off as the subjects became accustomed to swallowing the tube, which is reflected by the statistically insignificant individual variations in the volumes of gastric juice aspirated during the subsequent phases of the experiment. The variations in the hourly volumes were statistically insignificant. This corresponds with the results of Olson and Necheles.[34] The distribution and mean hourly volumes were in accord with those found by Olson and Necheles[34] and Kirsner and Ford,[35] and conformed to the expected normal variation.

The values obtained for gastric acidity agree with the statement by Donovan and Tighe[36] that the final concentration of free acid in gastric juice is never much higher than 50 mg. per litre.

Although the statistical analysis indicated no effect of vitamin C on hydrochloric-acid production, a significant skewness was revealed with the test of symmetry after the ingestion of 500 mg. of ascorbic acid. This skewness was perhaps due to the fact that some ascorbic acid remained in the stomach in spite of our efforts to remove it all.

Ascorbic acid, being an organic acid, formed part of the total acid value, but might also have affected the end-point in free-acid titration. In 5 cases treated with ascorbic acid the gastric juice was measured and titrated every 15 minutes, and revealed a relatively higher total acid content during the first and second 15 minutes of the experimental period than at any other time. This we regarded as support for the above explanation.

The obvious conclusion to be drawn is that too short a time was allowed for the vitamin to pass completely from the stomach into the intestine and that in spite of the aspiration at 10 a.m. some vitamin C remained in the stomach. With 1,000 mg. of ascorbic acid reasonable quantities of vitamin C were still left in the stomach after 1 hour, but none after 2 hours.

The results with 1,000 mg. of ascorbic acid clearly indicated that this level of the vitamin had no effect on hydrochloric-acid secretion after 2 hours.

Except for one male volunteer who excreted 35·1 mg. of total neutral 17-ketosteroids, the other subjects had 17-ketosteroid values which correspond closely with the results so extensively reported in the literature. A thorough statistical analysis of the ketosteroid-excretion, revealed no significant variation in its excretion with high doses of vitamin C.

DISCUSSION

Generally speaking, vitamin C, in the doses used in this experiment, had no effect on the volume of gastric juice secreted, nor the amount of hydrochloric acid produced. This is perhaps contrary to what one would expect, for, even if vitamin C has no direct effect upon the oxyntic cells of the fundus glands, the adrenal corticoids have a definite influence upon gastric secretion of hydrochloric acid,[14-19] and the adrenal glands, in their turn, contain relatively large amounts of cholesterol and ascorbic acid. The cholesterol and ascorbic acid of the adrenals markedly decrease and the cortical steroid hormones increase when the glands are stimulated by conditions of stress or injection of adrenocorticotropic hormone. These facts suggest that cholesterol serves as the precursor for the synthesis of cortical hormones and that ascorbic acid, in some unknown manner, is involved in this process.[37]

According to Ungar[6] vitamin C exerts a protective action on the stomach. If this is so, it is most probably a direct effect of vitamin C on the tissues of the gastric mucosa, for in the present experiment the vitamin did not affect either the volume or hydrochloric-acid content of the gastric juice secreted during the experiment.

From the previous discussion it is clear that the volunteers who took part in our experiment, may be regarded as 'normal' people. Admittedly the number of subjects was too small to justify the drawing of any definite conclusions; but statistically there were no indications that the results would have been at all different if 3 or 4 times as many subjects had been used.

It can therefore be stated that, under the conditions of this experiment, ascorbic acid exerted neither a beneficial nor an adverse effect on the gastric secretion or volume of gastric juice. Neither did the urinary ketosteroid excretion reflect any perceptible influence of ascorbic acid, in the doses given, on the adrenal cortical function.

The results of this investigation do not justify any explanation of the alleged beneficial effect of ascorbic acid in the treatment of gastric ulcer, and certainly not on the basis of an inhibitory effect of the vitamin on hydrochloric-acid secretion by the oxyntic cells.

The results and opinions expressed in the interesting article by Drummond[38] call for a repetition of this experiment under conditions where the tube is introduced on the evening before the experiment, and where the vitamin is administered parenterally, in a further effort to minimize all irrelevant influences.

SUMMARY

The effect of high doses of ascorbic acid (500 and 1,000 mg. respectively) on the secretion of gastric juice in normal healthy male and female students was investigated.

In additon the excretion of total neutral 17-ketosteroids and of 17-ketogenic steroids in the urine was determined, and used as a criterion for adrenal-cortical function.

Except for an increase in gastric secretion during the first control experiment, explained on phychosomatic grounds, there was no evidence that ascorbic acid exerted either an inhibitory or a stimulating effect on the secretion of gastric juice, or of hydrochloric acid.

Judging by the corticoid excretion in the urine, the adrenal cortex was not stimulated.

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UNUSUAL TREATMENT OF A POND FRACTURE

J. J. LOOCK, M.B., CH.B.

Queen Mary Hospital, Uitenhage

One not infrequently sees Pond fractures—and if one reads surgical text-books by different authors then one finds much difference of opinion as to whether and when these should be lifted. Perhaps there would be less controversy if the treatment were simpler. The following case illustrates how simple correction of the defect may be in some cases.

Some few weeks ago a 6-months-old Native baby was admitted to the Queen Mary Hospital, Uitenhage, suffering from a Pond fracture 1½ inches in diameter over the left occipital bone. This injury he had sustained at the heel of his uncle's boot! The patient's general condition was good and there were no abnormal neurological findings.

Despite the fact that the depression seemed to be causing no harm it was decided to lift it. The surgical

armamentarium consisted of (*a*) a scalpel, (*b*) a twist-bit used for drilling holes in wood, and (*c*) a cutting needle, needle-holder and 6 inches of dermalon.

For premedication he was given (*a*) phenergan, 25 mg. by mouth, (*b*) seconal, 1 gr. *per rectum*, (*c*) atropine, 1/200 gr. by injection.

It was originally intended to use local anaesthetic, but despite the fairly heavy premedication the patient was too restless and a gas and oxygen anaesthetic was given.

A vertical incision ½ inch long was made over the centre of the depression down to bone. Digital pressure on each side of the incision controlled all haemorrhage. The twist-bit was inserted through the incision and the starting thread was screwed into the skull till it was firmly fixed there. The depression was now easily elevated by exerting gentle traction on the twist-bit. One deep dermalon stitch closed the scalp wound and controlled bleeding. The entire procedure took 5 minutes.

The patient was fit for discharge only a few hours after operation.

Thanks are due to Dr. J. B. Smith, who administered the anaesthetic, the theatre staff for allowing the twist-bit in the sterilizer, and my father-in-law for the loan of the twist-bit. I also wish to thank the Medical Superintendent for allowing me to publish the case.

BAYTENAL ANAESTHESIA IN THE AFRICAN

REPORT OF 100 CASES

J. R. DUFFIELD, M.B., B.CHIR. (CANTAB.), D.A. (R.C.P. & S)

and

H. GINSBERG, M.B., B.CH. (RAND), D.A. (R.C.P. & S. I)

Baragwanath Non-European Hospital, Johannesburg

Most of the reports on Baytenal (the sodium salt of 5·5 allyl-methylpropyl-thiobarbituric acid) have come from Germany. Weese and Koss[1] described 350 cases, Vollmer and Haaf[2] 1,100, and Dietmann[3] 500. In the English literature the only articles we have found are by Nobes,[4] who reported its use in 11 cases, and more recently by Davidson and Love[5] who described 200 cases anaesthetised with Buthalitone sodium, the British equivalent of Baytenal.

The German writers compared it almost exclusively with soluble hexobarbitone. It was therefore thought that further clinical trials should be carried out to compare it with sodium thiopentone. Furthermore our patients were all non-Europeans and might be of interest on that account.

Physical Properties and Pharmacology

Baytenal is supplied as a yellow powder in 1-g. vials. Before use it is made up in a 10% aqueous solution. It is readily soluble in water, the solution being alkaline.

Previous work[1] has indicated that in dogs the safety factor is about 3. The dogs woke up without any excitement. According to experiments in rabbits, blood-sugar levels remained unchanged and there was nothing abnormal in the urine. On section the kidneys and other parenchymatous organs showed no histological change. Baytenal is broken down in the liver.

Clinical Findings

We have used Baytenal in 100 cases, all of which were in-patients, the majority undergoing minor gynaecological operations. It was given either alone or in combination with nitrous oxide and oxygen, supplemented in some cases with either minimal trichlorethylene or an ultra-short-acting relaxant. Premedication in most cases was with atropine, gr. 1/100. In our experience Baytenal has been more difficult to administer satisfactorily than sodium thiopentone, owing to a combination of delayed action (*nachhinkende Wirkung*[1]) and rapid elimination. When it was given at the same rate as thiopentone, this delayed action often led to an unnecessarily large dose being given. On the other hand, when it was given at the rate suggested by Weese[1] (1 to 3 g. at a time, with an interval of 1 to 1½ minutes before the next dose) the patient sometimes remained awake owing to rapid elimination.

Subsequent technique therefore followed fairly closely that of Nobes,[4] who gave a single rapidly injected dose of at least 0·5 g. for an adult. Having roughly estimated the required dose before injection, we gave 0·6 to 0·9 g., depending on the patient's physique, in a single dose. This was followed by nitrous oxide and oxygen or by small supplementary doses of Baytenal as required. Conducted in this way, the anaesthesia was in most cases satisfactory. There was only mild respiratory depression. The fall in blood pressure was comparable to that of ordinary sleep.

One pleasing feature was that in all cases abdominal relaxation was adequate in light planes of anaesthesia, comparing very favourably with that of sodium thiopentone at similar levels. There were, however, more side-effects than with thiopentone, some of them reminescent of soluble hexobarbitone. The commonest was coughing (31%). Hiccup occurred in 29% and laryngeal spasm in 3%. There was retching during induction in 2 cases and sneezing and jactitations each occurred in one case.

It is possible, that these reactions were more frequent

owing to the rapid rate of injection, but Davidson and Love[5], using Buthalitone sodium by 'crash induction' encountered hiccup in only 10% of cases and make no mention of cough.

The average dose given was 0·8 g. The criterion of emergence from sleep was the ability to respond when called by name; the average time after the last dose was 10¼ minutes. Once they had awakened, the patients appeared to be more wide-awake than after comparable doses of sodium thiopentone; 52% were able to move across on to the trolley 3 to 4 minutes after emergence.

The potency appears to be between one half and one third of that of thiopentone and it is possible that with comparably small doses of thiopentone patients would emerge equally early, but it has been our impression that the thiopentone patients, though awake, were more somnolent during the 3-4 hours after operation. This accords with Nobes' experiment on a healthy volunteer.[4]

Post-operative Complications

No post-operative nausea or vomiting were observed. These, however, are uncommon in the non-European after any type of anaesthesia. In 3 cases a small quantity of Baytenal was injected outside the vein but this did not give rise to any inflammatory reaction.

SUMMARY AND CONCLUSIONS

One hundred non-European cases anaesthetized with Baytenal are reported. From this preliminary trial it would appear, that Baytenal may be useful in the treatment of out-patients for minor procedures when early ambulation is particularly necessary. The indications and contra-indications are the same as for thiopentone.

Much more work seems necessary before one can assess whether, compared with thiopentone, the apparently slightly shorter duration of anaesthesia outweighs the greater difficulties of administration and higher incidence of side-effects, especially in view of the presence on the market of a new antidote to barbiturates (Megimide) at present undergoing clinical trials.

We wish to express our gratitude to the surgeons at Baragwanath Hospital for their patience and forebearance, to the members of our anaesthetic staff for their help, and to Messrs. Vernleigh Products for supplying the Baytenal.

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CHRONIC SUBDURAL HAEMATOMA: A CASE REPORT

H. H. LAUBSCHER, M.B., CH.B. (CAPE TOWN)

Rehoboth, South West Africa

When practising in a far-off town in South West Africa, with its vast distances, the lone general practitioner every now and again has to undertake surgery or some other procedure for which he is not at all qualified or in which he has had little or no experience—simply because the case cannot wait and will either die or sustain major disablement if what is needed is not done immediately and on the spot with such instruments as may be available. To illustrate this point the following description of a case of chronic (fluid) subdural haematoma is submitted:

CASE REPORT

On 23 December 1955 a Native (Nama) male, aged about 35, was brought in from a farm with a nasty septic wound 1½ inches long over the lateral half of the left eyebrow. He had been kicked by a horse about 4 days before and had been unconscious for about an hour. Afterwards he had a dull headache only and did not desire medical attention. When, however, the wound became septic he was brought in and admitted to the small local hospital.

He was found to have a grossly septic wound, 1½ inches long and running in the horizontal plane, over the outer half of the left supra-orbital ridge and extending towards the temporal region. There were no neurological signs other than a slight headache. The skull was X-rayed, but showed no fracture. He was put on antibiotics, the wound dressed daily, and he was discharged on the 10th day, fit and with the wound completely healed.

On 22 February 1956 I was summoned in haste to the farm where this boy was a labourer to come and see him. The farm was some 20 miles away over very rough roads. The patient was found to be deeply unconscious, although not comatose. The story was that he had been quite well up to 3 days before, when he started complaining of a headache, which became progressively worse; he then became confused and finally unconscious. He had then been unconscious for at least 18 hours. On the past and present history a preliminary diagnosis of chronic subdural haematoma, with compression of the brain, was made. The pulse was extremely slow, about 36 beats per minute, and the breathing, deep and sighing, was 7 per minute. The pupils were unequal in size. As a thorough neurological examination was impossible under the circumstances, and in any case was thought to be too time-consuming, he was immediately loaded on the back of a truck and literally bumped back to town over stones, ditches and corrugations. I stopped half-way to see whether he was still alive, and found him to be comatose; the pulse and breathing were still slower and the pupils were much larger and still unequal. He was given 2 c.c. of coramine, but it was thought that he would not last the rest of the way.

Surprisingly enough, when we reached the hospital his level of consciousness was much higher; he reacted to painful stimuli and even mumbled a few words when sufficiently aroused. The pulse was still very slow, while the pupils had become a little smaller. After a few minutes, however, he sank back into a coma again. There were no means of doing a ventriculogram or even a good enough X-ray to show up a collection of blood. There was in any case little doubt about the diagnosis or even the site of the haematoma, because of the previous history. Then came the question, what to do? If he was left alone, there was no doubt about the outcome; if taken to the nearest big town, over 60 miles of bad road—and where there was no neuro-surgeon—he would almost certainly die before he arrived. Therefore there was no alternative; a trephine operation had to be done right away. A quick survey of our instruments showed that all we had

in the line of 'brain surgery' were two old and very rusty trephines of the Horsley variety, ½ inch and ¾ inch in diameter. So what meagre instruments were available were assembled and the patient prepared for surgery. As he was already unconscious, an anaesthetic was unnecessary.

A cruciate skin-incision, each limb 2 inches long, was made in the left mid-temporal region, directly over the thickest part of the temporal muscle, and carried through the aponeurosis. The muscle was then split along its fibres to expose the skull covered by periosteum, which was scraped away to uncover a large enough area of bone for the trephine. The smaller trephine was first used and, although it was very blunt, a hole was eventually burred through the skull and the piece of bone removed. A cruciate incision was then made through the dura and the edges retracted by means of sutures. In the confined space a bluish membrane could just be detected. The larger trephine was then used to enlarge the opening through the skull, and the bluish membrane was opened. Fluid shot out under high pressure, first clear, resembling cerebro-spinal fluid, and then mixed with fluid blood, until a dark-red mixture, predominantly of fluid blood, came out. No suction, nor effective ways of irrigation was available, so large swabs were used to soak up the fluid, and as the flow diminished, smaller swabs were gently pushed into the cavity with forceps until enough was absorbed to show up the surface of the brain. It was then seen that the brain was fully 1 inch away from the dura and that the convolutions were very flat; it must have been under great pressure. Tepid saline was then poured into the cavity on to the brain surface and gentle swabbing was continued until the area was more or less dry. Blood clots were completely absent—it was all fluid. It was then noticed that the patient showed signs of returning consciousness; this progressed rapidly, and after a few minutes he started mumbling and had to be kept down. Unfortunately, no doubt owing to the tension and excitement, no check was kept on the patient's pulse.

A thick rubber drainage-tube, ¼ inch in diameter, was then inserted through the burr hole for a distance of ¾ inch and sutured into place. The dura was left open and the temporal muscle and superficial layers were sutured around the drain. By this time the patient talked and responded normally and wanted to know what was happening and what we were doing to him! Local anaesthetic was instilled before suturing the skin. A normal-saline drip was run in intravenously to aid expansion of the brain—500 c.c. at a fast rate and another 500 c.c. at a much slower rate.

By the next morning, i.e. 12 hours after the operation, the respiration, pulse and pupils were back to normal. The drain was inspected and completely removed after 24 hours. The patient made an uneventful recovery, the stitches were removed on the 7th day and he left the hospital on the 10th post-operative day, completely normal and very much annoyed about his clean-shaven head!

A point of interest is the considerable delay, i.e. 18 hours, that elapsed between his becoming unconscious and the time of the operation and, under such adverse conditions, the quick and complete recovery that followed. At the time of writing, which is 8½ months after the operation, the patient is still alive and well; in fact, it was reported to me by his employers that, if anything, he is now brighter than he was before, and his relatives have not noticed any changes in intelligence or personality.

SUMMARY

1. One is sometimes called upon to operate under the most adverse circumstances and on cases for which a surgeon specialist would normally be required.

2. A case of chronic (fluid) subdural haematoma, with compression of the brain, following on a head injury 2 months before, is described to illustrate these points.

3. A trephine operation had to be done immediately as a lifesaving procedure under the most adverse conditions, and by a team not at all trained in brain surgery, and yet in spite of this the operation was a complete success.

4. The patient had already been unconscious for 18 hours before the operation, but he made a very rapid and complete recovery without any residual neurological signs or symptoms. He was afterwards able to resume his former work, and his relatives have not noticed any changes in intelligence or personality behaviour.

POLIOMYELITIS VACCINE : STATEMENT BY THE MINISTER OF HEALTH

(By Our Parliamentary Correspondent)

The Department of Health is investigating the possibility of importing sufficient poliomyelitis vaccine from the United States to meet the Union's immediate demands, according to an announcement by the Minister of Health (Mr. J. H. Viljoen) in the House of Assembly on 29 January 1957.

The Minister made this announcement in the course of a reply to Mrs. S. van Niekerk (Drakensberg) who had asked what was the approximate number of doses of poliomyelitis vaccine for which applications were received by the Department of Health during 1956, how many doses were supplied, and whether there was a shortage of the vaccine. Mr. Viljoen said his Department did not deal with either the applications for or the issue of poliomyelitis vaccine, as these matters were handled directly by the South African Poliomyelitis Research Foundation.

'Unfortunately', he said, 'the Foundation has not been able to furnish me with the information asked for as the records of the Poliomyelitis Research Foundation are not kept in such a form that the information can be readily extracted therefrom'.

Dealing with the present position regarding the demand for and supply of poliomyelitis vaccine, the Minister said that when he made a statement to the Press on 5 December 1956, he indicated that sufficient supplies of vaccine would be available by the end of the year or early in the new year to meet all demands for first injections received until then. At the time the applications received amounted to approximately 450,000.

'By the end of the year 346,400 doses were in fact issued and the tests on a further batch of 80,000 doses were nearing completion', said Mr. Viljoen. 'In the statement referred to, I stressed that the strict control measures applied by my Department might, however, entail delays. Unfortunately, owing to unavoidable circumstances my Department has not yet seen its way clear to release this further batch, which is at present undergoing further tests.

'This, together with the increased demands for vaccine over the past weeks precipitated by the present high incidence of the disease, has therefore resulted in a shortage which, I trust, will only be temporary, as I am informed that the Poliomyelitis Research Foundation expects to double its output in the near future.

IMPORTATION OF VACCINE

'In the aforementioned Press statement I further indicated that licences for the importation of poliomyelitis vaccine from the United States had, on application, been granted to two commercial firms in the Union. I am informed that as yet no vaccine has reached this country through these channels. Therefore, in order to meet the shortage which has now arisen, my Department is itself investigating the possibility of importing sufficient vaccine to meet immediate demands.

'My Department will remain dependent upon the cooperation of the Poliomyelitis Research Foundation in regard to the distribution of whatever supplies of vaccine may become available through importation. Consequently all applications for poliomyelitis vaccine should continue to be made to the Foundation.'

NATIONAL COUNCIL FOR THE CARE OF CRIPPLES IN SOUTH AFRICA

CEREBRAL PALSY DIVISION

Since its inception in 1939, the National Council for the Care of Cripples has always dealt with a certain number of cases of cerebral palsy through its constituent Cripple Care Associations. Through the indefatigable work of its members and associates, the cerebral-palsy work has gained momentum and special activities, particularly schools for cerebral-palsy children, have been developed. The Pretoria school for cerebral palsy, operating within the orbit of the Pretoria and Northern Transvaal Cripples' Care Association was accepted by the National Council for the Care of Cripples as a pilot scheme in 1952, when the first of several grants of £1,000 was allocated from the Council's Easter Stamp Fund to the Cripple Care Association to assist in maintaining and expanding this school.

Great strides have since been made, culminating in the formation of a special Cerebral Palsy Division of the National Council for the Care of Cripples. The committee of this division includes a representative of each of the 3 cerebral palsy associations operating in Johannesburg, Cape Town and Pretoria and representatives of the Council's 9 constituent Cripple Care Associations who are especially informed or interested in cerebral-palsy work.

The National Council for the Care of Cripples is accepted by Government Departments as the national body dealing with cerebral palsy, and it controls practically all of the cerebral-palsy work throughout South Africa, apart from purely Government institutions, with which it maintains a close liaison.

PROGRAMME OF ACTION

At the first fully constituted meeting of the committee of the Division, the following ten-point programme of action was adopted as the immediate aim of the Division:

1. Unity of action between the various bodies in South Africa working on behalf of the cerebral palsied, and cooperation with the Government.

2. To make facilities for the diagnosis and treatment of cerebral palsy available in the main centres of the Union and South West Africa, and to offer all possible help to local communities in the establishment of schools, clinics, treatment centres, parents' groups and cerebral-palsy associations. Immediate attention is being given to Port Elizabeth, Windhoek and East London. A pamphlet will be prepared containing suggestions on how to start local projects for the cerebral palsied.

3. To educate the public about cerebral palsy and secure the interest and support of medical, educational and other agencies. A journal is to be published and a library established.

4. To consider the needs of ineducable cases and establish a recognized standard of educability.

5. To cooperate with bodies providing training for personnel—doctors, therapists, teachers etc., consider the appointment of orthopaedic technicians and visiting teachers.

6. To compile a national register of all cerebral palsied persons in South Africa and to cooperate with other organizations in this work.

7. To give attention to the needs of non-Europeans afflicted with cerebral palsy, a field hitherto almost untouched.

8. To carry out clinical and educational research into the causation and treatment of cerebral palsy, its prevention and its early diagnosis.

9. To establish hostels in large centres for children from country areas undergoing treatment and education.

10. To secure employment or sheltered employment for patients who need it, and to consider the foundation of a permanent Home for the very severely handicapped.

Achievements

The following achievements have so far been reached by the Division in cooperation with:

1. A cerebral-palsy school and treatment centre has been opened in Port Elizabeth by the Cerebral Palsy Division of the Eastern Province Cripple Care Association.

2. All the constituent associations of the National Council in areas where there is no cerebral palsy association, school or home, have set up special committees to carry out the Division's programme locally. As a result details are available concerning all the known cases of cerebral palsy in most areas.

3. The 3rd issue of the *South African Cerebral Palsy Journal* has now appeared.

4. A successful vacation course on the Education of Cerebral Palsied Children was held in Pretoria, for teachers in cerebral palsy schools. Over 100 teachers enrolled, from all over the country.

5. The *Natal Mercury* Children's Holiday Home Association will accommodate cerebral-palsy children at the Hibberdeen Holiday Home during school-going periods.

6. An offer of assistance has been made to the Rhodesian Society for the Blind and Physically Handicapped, 40% of whose cases are cerebral palsy cases.

7. A preliminary survey of known non-European cases of cerebral palsy in Johannesburg has been made.

Other Projects Pending

The following are further pending activities now engaging the attention of the Division:

1. Negotiations are proceeding with the United Cerebral Palsy Association of South Africa, an independent body who runs a clinic at Rosettenville, Johannesburg. It is hoped that these negotiations will bear fruit in closer unity.

2. Two projects are adumbrated for the setting up at non-European hospitals of clinics for the treatment of non-Europeans afflicted with cerebral palsy, one at Edenvale in Natal and the other in Johannesburg

3. The Transvaal Association for the Care of Cerebral Palsy is negotiating for the purchase of a property near its present school for use as a hostel for children brought in from the country for schooling and treatment.

4. Preparations in Natal continue for the establishment of a cerebral palsy rest home on the coast, and devoted work is being done in the raising of funds.

5. Definitions of cerebral palsy and of a brain-injured child, with special reference to South African conditions, are being drafted in consultation with various specialists and schools, to serve as a guide to schools and to the Department of Education, Arts and Science.

IN MEMORIAM

MARGARET ORFORD, M.B., CH.B., M.R.C.O.G.

Dr. A. J. Orenstein, C.B., C.M.G., C.B.E., of Johannesburg writes:
As one who has a cherished memory of being counted a friend by the late Margaret Orford, I beg permission to pay a short tribute to her memory.

Margaret had but a short life, and in many ways a hard life. Yet in only about a decade of specialist practice she built up a very high professional reputation. This is no mean achievement. It is, however, Margaret Orford, the woman of unassailable integrity, of profound loyalty and devotion to her profession, of remarkable courage and, above all, most lovable, whom I with her many friends now mourn and shall always remember. The mounds of flowers and the great congregation at her funeral attest the extent of respect and affection she so fully earned from patients, friends and colleagues. She died suddenly on 2 January

1957. It was the 'sudden clean death' she wished for herself, fearing that in her case it might be preceded by a long invalidism. That her wish was granted is some consolation to all who knew—and knowing, admired and loved—Margaret Orford.

MARGARET ORFORD MEMORIAL FUND

A group of lay friends of the late Dr. Margaret Orford, have approached certain members of the medical profession proposing the establishment of a Margaret Orford Memorial Fund by voluntary contribution without any public appeal. Brigadier A. J. Orenstein, Prof. James Black, Prof. G. A. Elliott, Dr. N. E. C. de la Hunt, Mr. Stephen Anderson and Mr. Cecil Payne have been asked and have consented to serve as a Board of Trustees to administer the Fund, which is to be used to assist deserving South African students of medicine.

MEDICAL ASSOCIATION OF SOUTH AFRICA : MEDIESE VERENIGING VAN SUID-AFRIKA

OFFICIAL ANNOUNCEMENT : FEDERAL COUNCIL : AMPTELIKE AANKONDIGING : FEDERALE RAAD

Notice is hereby given that a meeting of the Federal Council will be held at Medical House, 5 Esselen Street, Johannesburg, on 27, 28 and 29 March 1957, commencing at 9.30 a.m.

AGENDA

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the minutes.
5. Financial statement by Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from previous meeting.
9. Notices of motion transferred from previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin
Secretary

Medical House
Cape Town
1 February 1957

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word te Mediese Huis, Esselenstraat 5, Johannesburg, op 27, 28 en 29 Maart 1957, aanvang 9.30 vm.

AGENDA

1. Kennisgewing wat die vergadering belê.
2. Volmagte.
3. Notule van die vorige vergadering (reeds uitgestuur).
4. Sake wat uit die notule voortspruit.
5. Finansiële verslag van die Ere-Penningmeester.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae van vorige vergadering oorgehou.
9. Voorstelle waarvan kennis op vorige vergadering gegee was.
10. Nuwe kennisgewings van voorstelle.
11. Ander sake.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
1 Februarie 1957

PASSING EVENTS : IN DIE VERBYGAAN

The Nutrition Society of Southern Africa is holding its Inaugural Meeting on 11 February 1957, with a dinner at the Railway Restaurant, Johannesburg, and a subsequent meeting at Medical House, Esselen Street, Johannesburg. Professor Martha Trulson, Chief Nutritionist, Department of Nutrition, Harvard University, will give the Inaugural Lecture on 'Dietary Studies in Contrasting Population'. Further details can be obtained from Prof. J. T. Irving, Dental Research Unit, Oral and Dental Hospital, Milner Park, Johannesburg.

* * *

Mr. Cyril Kaplan, M.Ch.Orth., F.R.C.S. (Eng.), of 70-73 Trust Buildings, Gardiner Street, Durban, has been awarded a Fellowship for Advanced Study in Reconstructive Surgery and Rehabilitation by the Kessler Institute for Rehabilitation, Newark, New Jersey, USA. The programme includes study at special centres in the USA in addition to work at the Institute. Mr. Kaplan, accompanied by his wife, Dr. Sylvia Kisner, and children, will leave for America on 15 February 1957, and will be away for a period of 6 months. During his absence his practice and appointments will be conducted by Mr. F. J. Hedden, F.R.C.S. (Eng.), F.R.C.S. (Edin.), from the same address.

* * *

Prof. J. T. Irving, M.A., Ph.D., M.D., Director of the Dental Research Unit, University of the Witwatersrand and the South African Council of Scientific and Industrial Research, Oral and Dental Hospital, Milner Park, Johannesburg, has recently been elected an Honorary Life Member of the New York Academy of Sciences.

* * *

Oranje Vrystaat Provinsiale Hospitale. Kragtens artikel 358 van Administrateurskennisgewing nr. 63 van 1941 (O.V.S.) moet geneeshere formele aansoek doen om hulle private pasiënte in Provinsiale Hospitale te behandel. Die Uitvoerende Komitee het onlangs neergelê dat sodanige geneeshere voortaan persoonlik voor die Adviserende Rade van die hospitale vir 'n onderhoud moet verskyn, en aangesien hierdie prosedure as gevolg mag hê dat die magtiging van die Administrasie nie spoedig verkry kan word nie, is dit raadsaam om geneeshere op die vereistes te wys en aan te raai om vroegtydig vir die nodige toestemming aansoek te doen, anders kan dit maklik gebeur dat hulle ten koste van hul praktyk op die magtiging van die Administrasie moet wag.

* * *

Dr. John G. Cowley, M.B., B.Ch., D.T.M. & Hyg., M.R.C.P. (Edin.), has commenced practice as a Dermatologist at 27 Lister Buildings, Jeppe Street, Johannesburg. Telephone: rooms 22-5307, residence 42-5489, emergency calls 22-4191.

Dr. John G. Cowley, M.B., B.Ch., D.T.M. & Hyg., M.R.C.P. (Edin.), praktiseer nou as Dermatoloog by Lister-Gebou 27, Jeppestraat, Johannesburg. Telefoon, spreekkamers 22-5307, woning 42-5489, noodoproepe 22-4191.

* * *

World Health Day. Dr. F. J. C. Cambournac, Regional Director for the World Health Organization in Africa, announces that, as in previous years, World Health Day will be observed on 7 April 1957. The theme chosen is 'Food and Health', and in Africa it will be particularly devoted to nutritional deficiencies and malnutrition and generally to the importance of the relationship between diet and health.

This year the observance of WH Day will be sponsored not only by WHO, but also by the United Nations Food and Agriculture Organization (FAO). The Director suggests that the celebration may include press articles, lectures, radio talks, exhibits, etc. Documents on the subject are available for use, for instance, in connection with health services, schools and public institutions, and may be obtained at the Regional Office, P.O. Box 6, Brazzaville, A.E.F. (telegrams, Unisante Brazzaville).

Mr. Hugh Benjamin, F.R.C.S. (Edin.), of Johannesburg, will shortly travel by air to Boston, Mass., on a visit to the Lahey Clinic.

* * *

Die Christelike Geneesheersbond nooi alle geïnteresseerde dokters na 'n vergadering wat gereël is vir Woensdag, 20 Februarie om 8.15 nm. by die Karl Bremer-Hospitaal, Bellville. As sprekers sal optree dr. J. S. du Toit, President van die Mediese Vereniging van Suid-Afrika, gevolg deur ds. J. S. Gericke, Vise-Kanselier van die Universiteit van Stellenbosch, wat sal praat oor *Is God ook Geneesheer?*

* * *

The Medical Christian Fellowship has arranged a meeting to which all interested practitioners are invited, to be held at the Karl Bremer Hospital, Bellville, on Wednesday, 20 February at 8.15 p.m. Dr. J. S. du Toit, President of the Medical Association of South Africa, will speak, followed by the Rev. J. S. Gericke, Vice-Chancellor of the University of Stellenbosch, on *Is God ook Geneesheer?*

Mr. George Dall, M.Ch. (Cape Town), has joined Mr. Arthur Helfet, M.D., M.Ch. Orth. (L'pool), F.R.C.S., in orthopaedic practice at 808 Grand Parade Centre, Cape Town. Telephones: consulting rooms 3-2409; residence Mr. Helfet 6-8527, Mr. Dall 69-1915; if no reply 69-2924.

* * *

Medico-Surgical Cinema. La Presse Médicale offers prizes of 100,000 Fr. each and other awards for medico-surgical films. The films must be of 16 mm. size. Only amateur films are eligible, which must not be subsidized or produced by any laboratory or firm. The jury will consider the didactic value of the film as well as its cinegraphic quality. There are no restrictions as regards the character of the film, whether silent or with sound, in colour or not. The award will be announced in March 1957, and films should reach Paris before the end of February 1957. The address is The Secretariat, La Presse Médicale, 120 Boulevard Saint-Germain, Paris VIe.

REVIEWS OF BOOKS : BOEKRESENSIES

PHYSIOLOGY AND BIOCHEMISTRY

Textbook of Physiology and Biochemistry. Third Edition. By George H. Bell, B.Sc., M.D. (Glasg.), F.R.F.P.S.G., F.R.S.E., J. Norman Davidson, M.D., D.Sc. (Edin.), F.R.F.P.S.G., F.R.I.C., F.R.S.E. and Harold Scarborough, M.B., Ph.D. (Edin.), F.R.C.P.E., M.R.C.P. With a foreword by Robert C. Garry, M.B., D.Sc. (Glasg.), F.R.F.P.S.G., F.R.S.E. Pp. xii + 1068. Illustrations (some in colour). 60s. net + 2s. 5d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1956.

Contents: Foreword. Preface. 1. General Introduction. 2. Carbohydrates. 3. Lipids. 4. Some Physico-Chemical Considerations. 5. The Proteins. 6. Nucleotides, Nuclei Acids and Nucleoproteins. 7. Enzymes. 8. Biological Oxidations and Reductions. 9. Water and Minerals. 10. The Vitamins. 11. Energy Exchange. 12. Food and Nutrition. 13. Temperature Regulation. 14. Hunger and Thirst. 15. Mouth, Oesophagus and Deglutition. 16. The Stomach. 17. Digestion and Absorption in the Small Intestine. 18. The Large Intestine. 19. Intermediary Metabolism Methods of Study. 20. Carbohydrate Metabolism. 21. Fat Metabolism. 22. Protein Metabolism. 23. The Blood. 24. The Formed Elements of the Blood. 25. Blood Pigments. 26. Summary of Liver Functions. 27. Circulation. 28. The Heart. 29. Dynamics of the Peripheral Circulation. 30. Vasomotor Control. 31. Circulation through Lungs, Liver, Spleen and Brain. 32. Respiration. 33. Respiratory Function of the Blood. 34. The Kidney. 35. The Fluids of the Body. 36. Special Senses. 37. The Skin. 38. The Chemical Senses. 39. Vision. 40. Speech and Hearing. 41. Neurone and Synapse. 42. Muscle. 43. Autonomic Nervous System. 44. Nervous System. 45. Spinal Cord. 46. The Brain Stem and Postural Reflexes. 47. The Cerebellum. 48. The Diencephalon. 49. The Telencephalon. 50. Conditioned Reflexes. 51. The Endocrine Glands. 52. Reproduction. 53. The Pituitary Body. 54. Cell Division and Heredity. 55. Growth and Senility. Units and Measures with Conversion Factors. Standard Weights. Index.

Bell, Davidson and Scarborough is one of the few modern textbooks which attempt to cover in one volume the fundamentals of both physiology and biochemistry. This attempt to combine and integrate the teaching of two closely related sciences is highly commendable and the result is, on the whole, successful. The scope of the book necessarily precludes detailed treatment of either subject, but no major principle is neglected. The practice of illustrating the text with figures and plates from original articles in the literature adds a valuable historical background, and the short list of references at the end of each chapter is well selected as a guide to further reading.

The 3rd edition shows no major change from the pattern of its predecessors but there has been some re-arrangement of the contents, and increased emphasis is placed on fundamental physical chemistry. Recent advances are duly noted in the fields of carbohydrate and fat metabolism, structure of the protein molecule, control of pituitary function, cutaneous sensibility, and haematology. The descriptions of blood coagulation and of blood groups are perhaps over-simplified.

New illustrations include a number of excellent photomicrographs, some of them in colour and some taken by electron microscopy. Unfortunately the colour plate of blood cells, which appeared in previous editions, is now so pale that it gives little idea of the appearance of cells in a properly stained blood-film.

As in the previous editions, the format of the book is excellent, the illustrations clear, and typographical errors very rare. While the authors do not claim the book to be more than an introduction to physiology and biochemistry it does present in a concise and economical form most of what a medical student is required to know for the professional examination in these subjects. To the postgraduate student it offers a concise survey of current beliefs in physiology and biochemistry.

A.W.S.

STORY OF A MEDICAL PRISONER OF WAR

Stalag Doctor. By I. Schrire. Pp. 209. 13s. 6d. net. London: Allan Wingate (Publishers) Limited. 1956.

I have enjoyed reading 'Stalag Doctor'. It is a simple and straightforward account of a doctor's experiences among the prisoners of war during World War II and it is told by a Cape Town doctor who, in England at the outbreak, joined the R.A.M.C. and was in France at the time of the *débacle* in 1940. His insistence on the terms of the Geneva Convention and his attitude to his captors keep him moving from camp to camp, eventually taking him to the notorious Colditz Fortress, where he was confined when the war ended.

He gives a clear picture of the attitude of mind of prisoners of war, although he admits he is no psychiatrist, and he tells of the work which he and other medical officers were able to do for them.

The book contains some passages of rather sardonic humour, but generally it is a readable account of the memories and recollections of a difficult period in a man's life which time will probably never erase.

A.H.T.

VASCULAR TUMOURS

Tumors of the Cardiovascular System. By Benjamin H. Landing, M.D. and Sidney Farber, M.D. Pp. 138. Figures 138. $1.50. Washington: Armed Forces Institute of Pathology. 1956.

Contents: Introduction. Tumors of the Pericardium. Tumors of the Heart. Tumors of the Blood Vessels. Tumors of the Lymphatic Vessels.

This fascicle on Tumours of the Cardiovascular System forms part of the Atlas of Tumour Pathology published by the U.S. Armed Forces Institute of Pathology. The aim, therefore, is to illustrate clinical and microscopic appearances. The text is consequently kept short, and sometimes appears sketchy. Nevertheless, the essential features which are necessary for the diagnosis are in most cases clearly stated.

Although the emphasis is on the histology, clinical photographs are furnished for some lesions. One misses, however, photos of such well-known entities as glomus tumours, Kaposi's sarcoma and cirsoid aneurysm.

As the authors point out, much of the confusion in the classification and nomenclature of vascular tumours is due to the fact that

often more than one tissue is involved and that the differentiation between malformations, hamartomas and benign neoplasms of blood vessels is often impossible on purely clinical and anatomical grounds. The authors, therefore, have attempted to classify the lesions of the blood vessels and lymphatics primarily on the basis of microscopic appearances, which proves an equally difficult task.

The standard of microphotography and reproduction is outstanding. The microphotographs are representative and characterized by great clarity, giving beautiful detail of all the important features, which makes this a valuable book for reference.

R.H.G.

X-RAY DIAGNOSIS

Roentgen Signs in Clinical Diagnosis. By Isadore Meschan, M.A., M.D. with the assistance of R. M. F. Farrer-Meschan, M.B., B.S. (Melbourne, Australia). Pp. xx + 1058. Illustrations: 2,216 on 780 figures. $20.00. Philadelphia & London: W. B. Saunders Company. 1956.

Contents: 1. Background Fundamentals for Radiographic Techniques. 2. Protection from Roentgen Irradiation. 3. The Radiologist—His Functions and Terminology. 4. Introduction to the Radiography of the Skeletal System. 5. Fractures of the Extremities, Epiphyseal Separations and Dislocations. 6. Fracture Healing, Complications from Fractures and General Summary of Treatment Methods. 7. Congenital and Hereditary Abnormalities of the Extremities. 8. Radiolucent Bone Diseases of Multiple Extremities. 9. Radiolucent Bone Diseases of a Single Extremity. 10. Osteosclerotic and Hypertrophic Bone Diseases of the Extremities. 11. The Radiology of Joints. 12. Radiography of the Skull. 13. Diseases of the Facial Bones, Paranasal Sinuses and Temporal Bones; Space-occupying Lesions within the Cranium. 14. The Radiography of the Vertebral Column. 15. Introduction to Analysis of the Chest. 16. Roentgen Signs of Abnormality of the Diaphragm, Pleura and Thoracic cage. 17. Radiographic Differentiation of Pneumonic Diseases Characterized by Flat, Diffuse Shadows in the Lungs. 18. Nodular Lesions of the Lung Parenchyma. 19. Lesions of the Lungs Characterized by Increase in Linear Markings. 20. Lung Lesions characterized by increased Radiolucency in the Lung Fields. 21. Radiography of the Mediastinum (excluding the Heart). 22. The Radiography of the Heart. 23. Congenital Heart Disease. 24. The Value of the Plain Survey of the Abdomen. 25. Radiography of the Urinary Tract and Suprarenal Glands. 26. Radiography of the Gallbladder. 27. Upper Gastrointestinal Tract: Oropharynx, Laryngopharynx, Esophagus. 28. Stomach, Duodenum and Pancreas. 29. Radiology of the Small Intestine. 30. Radiography of the Colon. 31. Radiography in Obstetrics and Gynaecology. Index.

Starting with the fundamental concepts in electricity and magnetism the author then describes the manner in which an X-ray machine operates, giving details about cassettes, intensifying screens, films and cones. He also describes the various types of X-ray machines for different requirements.

The effects of radiation on the human body are discussed, and methods of protection for the patient and operator described.

A chapter is devoted to the functions of the radiologist and the composition of the consultation report.

The authors then proceed to deal with the various systems of the body. Each chapter includes radiographic technique and positions.

The chapter on fractures includes lining techniques to illustrate the normal relationship of the bones for proper function of the joints. The methods of treatment for fractures is also described from the radiological standpoint.

The book is profusely illustrated with X-ray negatives and line drawings. The line drawings in particular are amongst the best yet seen by the reviewer. There is a full description with each drawing illustrating the essential features of the disease process.

This is a very useful reference book for specialists, students of diagnostic radiology, and medical students.

H.C.P.

RHEUMATISM

Der Rheumatismus. Herausgegeben von Prof. Dr. R. Hopmann, Köln. 100 Seiten. 29 Abbildungen. DM 16.50. Stuttgart: Georg Thieme Verlag. 1956.

Inhalt: Vorwort. 1. R. Hopmann: Einführung. 2. A. Goebel: Die Patologie des Rheumatismus (Mit 4 Abbildungen). 3. O. Guthof: Bakteriologie und Serologie des Rheumatismus. 4. H. Ewerbeck: Rheumatische Erkrankungen im Kindesalter (Mit 3 Abbildungen). 5. L. B. Seiferth: Die Bedeutung der Tonsillen und der Nassennebenhöhlen für die Herderkrankung (Mit 3 Abbildungen). 6. K. Fr. Schmidhuber: Odontogene Herde als mögliche Ursache von Fernwirkungen. 7. B. Schuler: Rheumatismus aus der Sicht des Internisten (Mit 5 Abbildungen). 8. M. Hackenbroch: Haltungsstörungen, degenerative Skeletterkrankungen und ‚Rheuma' (Mit 9 Abbildungen).

This booklet is a collection of short articles by individual writers and not a book from the pen of a single, experienced rheumatologist for the instruction of the practitioner.

There was a period when many books by German authors were pioneering efforts. This cannot be said about this 'nut-shell' publication on such an important practical subject. The happenings in Germany since the first world war caused an upset also in the German medical profession, while during the same period the British school of rheumatology, followed by others, came to the front.

This booklet, which has no index, lists many publications in English amongst the 'literature' at the end of each chapter. It is surprising that only a short classification of rheumatic disorders as used in Germany is mentioned, though the much more elaborate British list is well known in Germany, as are scores of other classifications. To speak about 'rheumatism' as a general term does not bring any new idea useful to the reader. Nor does the booklet contain anything that has not been published in English on one side of the Atlantic or the other. The old German nomenclature, so puzzling to the British school, is retained and the well-known findings of the great German workers in rheumatology (Klinge, Aschoff, etc.) repeated.

Beyond the statement that 'the conception of rheumatism is not as yet clear' (on page 8), I do not find much merit in this book. It is published in a language only few English-speaking doctors understand and read.

N.F.

HISTOPATHOLOGY OF THE SKIN

Histopathology of the Skin. Second Edition. By Walter F. Lever, M.D. Pp. 518 + xviii, with 281 illustrations. 96s. London, Philadelphia, Montreal: J. B. Lippincott Company. 1954.

This book was reviewed in the *Journal* of 24 November 1956 (**30**, 1149). The Pitman Medical Publishing Company Ltd., 45 New Oxford Street, London, W. C. 1., have acquired the right to supply Lippincott books in the South African market. They republished Lever's *Histopathology of the Skin*—Second Edition—in April 1956 and it is supplied at the price of 80s. net.

HEREDITY AND LOCAL DISEASE

Eredita E Localizzazioni Morbose. By Teodori-Borghi-Neri Serneri. Pp. 246. £.2000. Pisa: 'Omnia Medica'. 1955.

Contents: Introduzione. I. Importanza dell'eredita nel concetto di costituzione e di diatesi. II. Fattori ereditari dello 'stato disrafico' come causa di malformazioni variamente localizzate. III. Le asimmetrie genotipiche come fattore di localizzazione di processi morbosi. IV. Fattori genetici di localizzazione delle malattie infettive. V. Fattori ereditari della diatesi disreattiva edemato-pomfoide (cosidetta allergica) con particolare riguardo ai fattori condizionanti le sue localizzazioni. VI. La predisposizione genetica alle malattie reumatiche e alla loro localizzazione (reumatismo acuto primario e reumatismo cronico primario). VII. Aspetti genetici di localizzazione dei tumori. Riassunto. Bibliografia. Sintesi conclusiva.

The authors, all members of the University of Florence staff, tackle the stiff problem of the influence of heredity on the localization of morbid processes.

In the introductory chapter the authors confess their difficulty in defining the term 'constitution' in its medical connotation. In this volume they limit the term to individual, as opposed to social and national, characteristics. In this sense two main types of constitution exist, the phenotype and the genotype. The phenotype is descriptive of a class of organisms the members of which cannot be distinguished from one another, the genotype of a class wherein distinctions can be demonstrated by breeding tests. The authors examine at some length the effect of acquired characteristics as opposed to hereditary factors in determining the final structure and personality of the individual. While not accepting either the genotype or phenotype as expressive or explanatory of all the difficulties encountered, they offer their own conception.

The main portion of the book is devoted to an inquiry into the hereditary factors operative in certain specified fields of pathology. Here the case histories of twins, uniovular and binovular, yield an important contribution. First of all, there is the large field of congenital malformation, those due in the first place to non-closure of the primitive neural cord. The inherited tendency or constitution accompanying these deformities is termed the status dysraphicus. The list is a very long one and the authors include such conditions as neurofibromatosis, abnormal length of the arms and pigmentary anomalies of the iris with anisocoria as conditions which are closely

linked with an inherited constitution. When lesions like spina befida are present they suggest that these deformities are transmitted by a dominant gene of varying degree of penetration. Conditions like exstrophy of the bladder, undue length of the arms etc. they explain as the result of additional extragenetic factors.

The instances cited show that agreement is by no means universal whether the particular gene is a dominant or a recessive in any particular case. A striking coincidental occurrence of xanthoma, coronary sclerosis and hypercholesteraemia is revealed in the families studied. Xanthoma represents the homozygotic influence in heredity; hypercholesteraemia is a heterozygotic manifestation in a phenotype.

Other chapters examine the evidence for inherited factors in the localization of certain infectious disease; for example, the tendency to tuberculous disease of the kidneys or endocardial involvement in rheumatic disease. Observations and studies of certain family histories confirm what has indeed been common knowledge in certain circles, that not only is a predispostion to certain pathological states inherited, but a tendency for those states to be localized in certain organs. Of recent years the conviction has been steadily growing in the laity that cancer is a hereditary disease, and not only cancer generally but cancer specifically related to a certain organ, e.g. cancer of the stomach, the uterus or the breast. The authors record an impressive series of investigations—impressive both in extent and in the results that emerge. They quote numerous investigators who found that agreement in the localization and histological characters of tumours occuring in twins was 64% in monozygotic twins as against 5% in dizygotic, and they conclude that the predisposition to cancer is frequently but not always inherited. The inherited transmission is usually in accordance with Mendelian laws, though it is impossible to exclude cytoplasmic inheritance particularly in tumours of the female genitalia.

The book is really a collection and critical examination of the vast amount of investigation that has been carried out in this field. Conflict of views is frequent so that dogmatic statements are rare. But the extensive lists of references at the end of each chapter bear witness to the diligence and enthusiasm which the authors have brought to their labours. The nomenclature peculiar to the science of heredity is puzzling to the unitiated or casual reader. Some words of advice may be offered to the latter. Brush up your Italian by all means if you should be tempted to read this book. But in addition, polish up your knowledge of heredity and its terminology!

C.K. O'M.

CORRESPONDENCE : BRIEWERUBRIEK

CHIROPODY

To the Editor: I am happy to inform you that at an inaugural meeting held at Johannesburg General Hospital on 13 January 1957 the South African Branch of The Society of Chiropodists, England, was formed.

All members of the Branch are registered medical auxiliaries, and it is hoped thus to represent qualified chiropodists in South Africa.

C. B. Sroka
For the South African Branch of The Society of Chiropodists, England

504 African City
100 Eloff Street
Johannesburg
23 January 1957

THE POSITION OF HYDROTHERAPY IN SOUTH AFRICA

To the Editor: For some time I have been contemplating another letter on a subject which ought to be of interest to the profession, viz. treatment by water, or hydrotherapy. (The term 'crenotherapy' is used to denote all forms of treatment which may be given at a spa.[1])

The occasion to write again presented itself recently. The *East London Daily Dispatch* on 15 December 1956, published an item of news 'Baths Opened'. In the caption under a photograph it informed the readers about the 'hot sea-water baths' opened by the Mayor, who is seen to be assisted in this ceremony by the City Engineer, the Water Engineer and the Beach Manager. The caption told the reader that 'the sea-water baths are popular with invalids, who can enjoy the benefits of salt water in privacy and *at a temperature to their liking*' (the italics are mine).

My first reaction to this news was amazement. If this institution is to be used 'by invalids' why was the M.O.H. not connected with the official opening? Even if *tres faciunt collegium* I must doubt if the three gentlemen in this picture can advise an invalid on hydrotherapy. I have visited the establishment and the Beach Manager was good enough to conduct me through it; he definitely disclaimed any ability to give advice on the use of these baths.

My own considered opinion is that the baths, in their present form, should not be recommended for invalids, and I strongly protest against the dangerous information—which at first may appear to a layman to bear the approval of the local authority—that invalids can safely be left to choose what temperature they please for salt-water baths.

Why these baths should become popular is not stated. If used intelligently, under expert medical supervision, they serve a useful purpose. Indeed, I supported the proposal that these baths should be provided in a letter I wrote to the newspaper on 26 September, because I know from my observations in the past what such establishments offer to an invalid. But my support was in principle only, and I specifically added 'even if many improvements are actually essential'.

To allow the invalid to take hot salt-water baths without expert guidance is unscientific and dangerous. It smacks of the quackery practised at almost all our so-called 'spas' as evidenced in their brochures to the public. I have seen many bad results from such 'cures'.

Dr. Simon Baruch was the father of scientific hydrotherapy in the USA, where similar conditions prevailed. In his book[2] he stated: 'It is to be regretted that physicians in general do not place the estimate upon hydriatric treatment which it deserves, for with the exception of diseases of the nervous system (in which the water treatment is often used in a most senseless manner) little and bad use is made of it in diathetic affections'.

In the treatment of chronic diseases in general and of the so-called 'chronic rheumatic' group of diseases in particular, baths have been used for centuries past with great benefit to the sufferers, not only at mineral water springs, but also in institutions using only ordinary water or, at the coast, sea-water. I must emphasize that it is *chronic diseases*, not acute or subacute cases of the so-called 'rheumatism', that I am writing about.[3–5] The use of water in the treatment of various diseases is one of the ancient practices. We find reference to its use by Hippocrates.[6,1] The contra-indications of hot-bath therapy are organic diseases of the central nervous system, myocardial weakness, cardiac hypertrophy and arteriosclerosis.[1] The temperature of the water is of primary importance and it cannot be left to the fancy of any layman to use it 'to their liking'. Not only the temperature of the water but also the duration of the bath must be prescribed for an invalid.

I am sorry that all my efforts in this direction in the past[6–8] including my memorandum[9] to and evidence[10] before the National Health Services Commission brought no result. I will not claim that the hospital at Warmbaths, Transvaal, was the result of my papers and evidence. I had the honour to be appointed to the Board of this 'non-acute Hospital' from its inception in 1948 and served on it until my resignation in 1955, when I left Johannesburg for the coast. It was disappointing to me that hydrotherapy, which should have been the foundation of this special hospital, was relegated to the bottom of the list of treatments used. I am therefore perturbed to think that the same old methods and system of therapy, about which I was complaining,[1] will be continued in the hot sea-water baths at East London. I will therefore offer no apology for writing on this subject, which is still the stepchild of therapy in South Africa. I hope that real interest will be taken in future to put hydrotherapy on a sound basis here, just as overseas.

I have suggested[6] that 'The Government should encourage such work, to be undertaken by an Association of South African Spas to be established for the development of our springs in a modern way on scientific foundations'. In the fight against 'chronic rheumatism and allied conditions' we cannot do without hydro- or balneotherapy. One must agree with the sentiment in the leader of our *Journal* dated 23 July 1938: 'The initiative in this matter should be the task of our profession and should not be left to the Union Department of Public Health'.

Nathan Finn

27-30 C.N.A. Building
East London
31 December 1956

1. Finn, N. (1941): S. Afr. Med. J., **15**, 229.
2. Baruch, S. (1892): *The uses of water in modern medicine*. Detroit, Mich.: George S. Davis.
3. Finn, N. (1937): S. Afr. Med. J., **11**, 878.
4. *Idem* (1938): *Ibid.*, **12**, 487.
5. *Idem* (1938): Acta rheum. (Amst.), December.
6. *Idem* (1941): S. Afr. Med. J., **15**, 483, and (1942): Brit. J. Phys. Med., July-August.
7. *Idem* (1942): *Ibid.*, **16**, 230.
8. *Idem* (1943): *Ibid.*, **17**, 24.
9. *Idem* (1943): *Memorandum on Health Resorts and Spas*. Report of National Health Services Commission, Pretoria.
10. *Idem* (1943): Evidence on Rheumatism and on Spas, National Health Services Commission, Pretoria.

DOCTORS' RIGHT TO DISPENSE

To the Editor: The threat to deprive doctors of their ancient right to dispense medicines for their patients has not been abolished.

The great benefits bestowed on the public and the advantages accruing to the medical profession as a result of doctors dispensing for their patients have already been stressed.

Among other things, let it not be forgotten that the ten million Natives in South Africa have been able to pay for medical examinations and medicines on account of the noble and generous practice of doctors examining and treating them at very low charges. Should doctors be deprived of their right and duty to dispense for such patients, a great and unbearable burden is going to be placed on them.

For the sake of our patients, ourselves and the interest of medicine itself, let us not allow ourselves to be deprived of our right and duty to dispense.

Dispensing Doctor

25 January 1957

INFANT SUGAR URINALYSIS

To the Editor: The procuring of urine specimens in infants, is often a difficult and clumsy procedure. Occasionally unnecessary catheterization has to be resorted to, or the plausable remark 'specimen unobtainable' entered on the record sheet.

For this reason, I thought your readers might be interested in the simple method of Infant Sugar Urinalysis by means of 'Tes-Tape' (Lilly).[1] A 2-inch length of this tape is placed in, or affixed to, the baby's nappie and the test read as soon as the usual signs become evident.

This tape gives very reliable results; and although I do not claim this procedure to be necessarily original, nor particularly ingenious; it may help solve some colleagues' diagnostic difficulty.

J. Dommisse

Williston
Cape
24 January 1957

1. New Preparations and Appliances (1956): S. Afr. Med. J., **30**, 481.

‚DOKTER' OF ‚DOKTOR'

Aan die Redakteur: Na aanleiding van die skrywe[1] van dokter, doktor of meneer W. Steenkamp wens ek hom net te wys op die spelling soos aangegee in die Afrikaans-Engelse woordeboek: (1) Dokter—doctor physician; (2) Doktor—doctor (literature etc.).

Ons word dus dokters met die *e* en nie doktors met die *o* genoem nie. Ongelukkig kan aan die uitspraak nie verander word nie.

C. Bouwer

Posbus 13
King William's Town
22 Januarie 1957

1. Steenkamp, W. (1957): S. Afr. Med. J., **31**, 64.

THE 'MR.'-'DR.' PROBLEM

To the Editor: It seems that there is continuous confusion about the 'Mr.'-'Dr.' designation.

Some years ago I read in a historical volume that this issue began in Ireland, when surgery passed from the barbers to scientifically trained individuals. The physicians claimed and temporarily held the monopoly of the 'Dr.' The surgeons, later allowed the right to this appellation, now refused it, saying they did not wish to be confused with the 'lesser trained' physicians, and preferred the plain 'Mr.'

I discussed this with the late Mr. T. Lindsay Sandes—no mean student of history—who corroborated the record, adding that any surgeon trained outside the Land of the Shamrock, would merely be gate-crashing a purely Irish tradition if he preferred 'Mr.' to 'Dr.'

G. P. Fourie

314 S.A. Mutual Buildings
Cape Town
26 January 1957

ELI LILLY MEDICAL RESEARCH FELLOWSHIP (SOUTH AFRICA)

To the Editor: Applications for the 1957 award of this Fellowship may now be submitted and must reach the undersigned not later than 30 April 1957. The detailed conditions governing the award of this Fellowship appear in the 2 February 1957 issue of *Medical Proceedings* at p. 66. They may also be obtained from the undersigned.

H. A. Shapiro, Ph.D., M.B., Ch.B., F.R.S.S.A.F.
Honorary Chairman
Selection Committee, Eli Lilly Medical Research Fellowship (South Africa)

P.O. Box 1010
Johannesburg
21 January 1957

POLIOMYELITIS IMMUNIZATION

To the Editor: Is it not timeous to seek the reason for the marked rise in the incidence of poliomyelitis? We are aware of only one relevant factor that has been subject to variation, namely that there has been a piecemeal inoculation of small sections of the population with treated virus. The coincidence is too marked to ignore the possibility that we may have thereby disturbed the balance of nature and have, albeit unwittingly, brought about an increase in the 'pool' of carriers. The practice of administering gamma globulin to contacts may also be aggravating this unhealthy tendency.

The uninoculated members of the population appear to me to be in a precarious position, which leads me to suggest that it behoves G.P. groups to consider two burning questions: Is it not, at the present stage, advisable to recommend the suspension of further piecemeal and private inoculations; and to ask that a few of the Foundation's precious monkeys be rather used for the testing for infectivity of those contacts who have been inoculated in the past?

I have left much unsaid for the sake of brevity but cannot miss the opportunity of complaining about the lack of information available to the G.P. about the vaccine.

M. P. Freedman

156 Voortrekker Road
Goodwood
24 January 1957